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# AUTONOMIC AROUSAL AND SUBJECTIVE REPORT OF ANXIETY AND BEHAVIOR UNDER STRESS

by

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Dissertation submitted in partial fulfillment of
the requirements for the degree of Doctor
of Philosophy in the Department of
Psychology in the Graduate School
of Duke University

1973

# ABSTRACT

(Psychology-Physiological)

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# ABSTRACT

## AUTONOMIC AROUSAL AND SUBJECTIVE REPORT OF ANXIETY AND BEHAVIOR UNDER STRESS

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Three independent variables relating to autonomic arousal were studied as to their effect on subjective report of anxiety and overt behavior under stress. These independent variables were actual autonomic arousal, induced cognitions of degree of autonomic arousal, and attribution of that arousal to an emotional (intrinsic) or nonemotional (extrinsic) source. These variables had been investigated previously by Schachter and Singer (1962), Ross, Rodin, and Zimbardo (1969), Valins and Ray (1967), and others; but their data were not conclusive evidence for the hypotheses of interest, and no study had included all three of these independent variables previously. Interactions were predicted, specifically that high actual arousal or information that one is aroused and an intrinsic attribution for the arousal lead to higher subjective report of anxiety and to more behavioral disturbances and avoidance than are found when either of those conditions is absent. In addition, it was hypothesized that

information that one is unaroused produces less reported subjective anxiety, behavioral disturbances, and avoidance than no information in the presence of an intrinsic attribution for arousal. The effects for cognitions of arousal were hypothesized to occur without corresponding changes in actual autonomic activity.

The research was conducted in an oral surgery clinic, using subjects who were about to undergo extractions as subjects. Actual arousal was manipulated with the local anesthetic injection, which contained epinephrine for one group and no epinephrine for the other group. Cognitions of arousal were manipulated simply by telling the subjects that they were aroused or unaroused or, for purposes of comparison, by giving them no information. To induce an extrinsic attribution of arousal, subjects were told that the drug produced arousal; for the intrinsic attribution of arousal, subjects were told nothing about the source of arousal.

The predicted interaction of actual arousal and attribution was found only for males on the subjective report measure. Telling subjects that they were aroused interacted as predicted with attribution for both females and males on the subjective report measure, but subjects who were told that they were unaroused evidently did not accept this information as veridical. Manipulations of cognitions of arousal were not accompanied by changes in pulse rate or blood pressure. These results support a cognitive interpretation of the effects of autonomic arousal on subjective anxiety; both cognized arousal (either due to perception of actual arousal

or to induced cognitions of arousal) and the availability of only an intrinsic attribution for that arousal seem necessary for subjective report of anxiety. On the other hand, the manipulations generally did not affect the behavioral measures significantly. This difference is explained in terms of behavioral consequences which were unrelated to the subject's inference based on his cognized arousal and its attribution to an intrinsic source.

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# INTRODUCTION

The present era is so dominated by threats to the existence and happiness of the society and the individual that its identification as the "Age of Anxiety" has become a popular cliché. Concern with anxiety is not confined to the man on the street; behavioral scientists from diverse backgrounds spend innumerable hours in heterogeneous behaviors ranging from investigating differences between psychiatric and "normal" populations to traumatizing subjects with painful electric shocks--all in an attempt to understand, and presumably to alleviate, anxiety.

While the amount of empirical research on anxiety has increased greatly since 1950 (Spielberger, 1966), the utility of this research has been limited by the overinclusiveness of the term "anxiety," a term which lacks a common, invariant meaning, as pointed out by Mandler and Kessen (1959), among others. Researchers often have overgeneralized their findings to situations and procedures which imply radically disparate definitions of anxiety. Others have turned their attention to the nature of anxiety, involving themselves in esoteric definitional controversies.

Fortunately, we can extricate ourselves from the difficulties inherent in the question, "What is anxiety?"--simply by considering instead

the obvious, accessible, and important relationships among what have been considered as measures of anxiety. For our purposes, anxiety and fear can be equated, and we will consider, almost exclusively, measures of transitory or situational anxiety. We can deal with these variables as data in their own right rather than as measures of something else (anxiety). The present investigation takes that approach--it examines the effect of one common "measure" of anxiety, autonomic arousal,[1] on two other conventional measures, reported subjective anxiety and overt behavior in stressful situations. The main considerations will be whether autonomic changes play a causal role with respect to the latter two variables and, if so, the nature of the relationships. The particulars of these

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[1] The autonomic nervous system innervates cardiac muscle, smooth muscle, and glands. Cannon (1929) elucidated an "emergency reaction" and argued the functional value of these physiological reactions in "fight" or "flight." The modern equivalent is autonomic arousal, a change in autonomic activity toward sympathetic dominance. The elements listed by Wolpe (1958) would be widely accepted as autonomic arousal: "tachycardia, raised blood and pulse pressures, hypernea, pilo-erection, mydriasis, palmar hyperhidrosis, and dryness of the mouth (p. 35)." He also includes the parasympathetic effects of defecation and urination which occur in extreme cases, and which may be compensatory to sudden sympathetic outflow (Morgan, 1965). Many of the autonomic effects can be measured in intact humans with the appropriate apparatus. The most common measures include heart rate, blood pressure, peripheral vasoconstriction, respiratory rate, palmar sweating, and electrical resistance of the skin which is somewhat related to sweat gland activity. Although many of these variables are influenced directly or reflexly by skeletal muscle activity as well, the term "autonomic arousal" will be used throughout this paper because of the prevalence of the term in the anxiety and stress literature. The broader term, "physiological arousal," will be used less often, in order to prevent confusion with central nervous system measures of arousal or activation.

relationships obviously have important implications for therapeutic techniques and for diminishment of the deleterious effects of stressful situations.

## Is Autonomic Arousal a Sufficient Cause?

Traditional peripheral theories of emotion claim that changes in peripheral effectors, especially those controlled by the autonomic nervous system, are both necessary and sufficient for subjective emotion (James, 1884, 1890, 1894; Lange, 1922) or overt emotional behavior (Wenger, 1950a, 1950b). Beginning with a series of damaging criticisms of the peripheral view, Cannon (1927, 1929, 1931) advanced the alternative position that subjective emotional feeling and emotional behavior are centrally determined and independent of peripheral physiological changes. Although the peripheral theories have been vigorously defended (Fehr & Stern, 1970; Mandler, 1962; Schachter, 1966; Wenger, 1950a), most physiological theories subsequent to Cannon's criticisms have emphasized central nervous system mechanisms presumed to mediate subjective emotion and emotional behavior. Nearly all contemporary theories which do emphasize the contribution of peripheral autonomic activity also include central controlling factors, which may be described in neural or cognitive terms (Arnold, 1950, 1960, 1967; Bull, 1951, 1962; Schachter, 1966; Schachter & Singer, 1962). In other words, they consider the autonomic changes as contributory but not sufficient for subjective emotion and emotional behavior.

One notable exception is Wolpe's (1958) explanation of systematic desensitization; along with Schachter's theory, which is discussed in a later section of this chapter, Wolpe's technique of behavior therapy and his assumptions concerning desensitization account for much of the recent interest in autonomic arousal as contributing to subjective report of anxiety and maladaptive behavior under stress. Wolpe assumes that predominantly autonomic responses arising from noxious stimuli are felt by the individual and are unpleasant, and that it is *solely* these responses which lead to the subjective report of anxiety. In addition, the individual tends to avoid any situation in which these autonomic responses occur.

In systematic desensitization the individual visualizes a stressful situation while in a state of muscular relaxation. Wolpe explains the reduction of reported subjective anxiety and phobic avoidance which occur in successful desensitization as the *direct* result of reduction in the relevant autonomic responses. The logical extension of Wolpe's explanation has been suggested by others (Ascough & Sipprelle, 1968; Lang, 1970) in the form of a therapeutic refinement--operant conditioning or training in voluntary techniques as a more specific and powerful means of reducing autonomic arousal.

Granted, efforts to reduce the subjective anxiety reported and the maladaptive behavior exhibited in stressful situations are commendable, but the most efficient control of these two variables in the case of a phobia or any situation which is threatening depends on the actual direction,

strength, and exclusivity of the autonomic-subjective and autonomic-behavioral relationships. In addition to the original criticisms of the peripheral theories, several lines of evidence indicate that autonomic changes do not control subjective report or behavioral variables directly.

One preliminary type of evidence comes from studies which measure the covariance of autonomic variables with subjective report or behavioral variables under stress. Evidence that these variables are related or correlated would not necessarily indicate a controlling function for autonomic variables; on the other hand, a lack of correspondence would cast serious doubt on the controlling nature of autonomic variables. Although many researchers who intend to measure anxiety choose one dependent measure and assume that their study can be compared to experiments using other measures, the practice is not well founded. Recently, several writers (Buck, 1971; Lang, 1969; Lazarus, 1968; Mathews, 1971) have commented on the low correlations between autonomic, subjective, and behavioral variables measured in the same stressful situation.

Studies which have tested autonomic-subjective and autonomic-behavioral relationships have used diverse paradigms and measures, and many are open to methodological criticism; however, a brief summary is in order since the lack of correspondence is discussed rather than documented in the articles just cited. Only those studies using situational subjective report measures or situational measures of stress-related overt behavior are included.

Autonomic changes, usually in the direction of sympathetic dominance, are consistently found in experiments in which subjects are threatened (Martin, 1961). In several studies, subjects who reported high fear or anxiety also exhibited significantly higher autonomic arousal to stress than other subjects (Grossberg & Wilson, 1968; Hodges & Spielberger, 1966; Pearson & Thackray, 1970; Raphelson, 1957; Wilson, 1967). On the other hand, no significant difference was found in other studies (Katkin, 1966; Martin & McGowan, 1955; Rosenstein, 1960). Subjects who reported extreme fear of a situation also displayed significantly higher autonomic responses to relevant than to neutral stimuli in at least three desensitization studies (Geer, 1966; Marks, 1971; Van Egeren, Feather, & Hein, 1971); trials on which subjects indicated subjective anxiety were significantly higher than other trials for only some of the autonomic measures and only when one-tailed tests were used in a fourth study using a systematic desensitization analogue (Waters, McDonald, & Koresco, 1973). Significant autonomic-subjective correlations have been reported in several studies (Harleston, Arey, & Smith, 1965; Hodges & Spielberger, 1966; Katkin, 1965; Mordkoff, 1964; Pillard, Carpenter, Atkinson, & Fisher, 1966), but these correlations were generally quite low. Moreover, Marks (1971) found no significant correlation between autonomic arousal and subjective report during phobic imagery, and Weinstein, Averill, Opton, and Lazarus (1968) reviewed six studies in which correlations showed little agreement between autonomic and

subjective report measures. Proctor[1] found a rank-order correlation of nearly zero between pulse rate elevation and reported subjective anxiety in 21 patients awaiting oral surgery. In addition, several studies found incongruous changes in autonomic and subjective responses; although these relationships were not tested directly, obvious inconsistencies were found in the level (Hodges, 1968; Rappaport, 1971; Van Egeren et al., 1971), direction (Clemens & Selesnick, 1967), or timing (Epstein & Fenz, 1965, together with Fenz & Epstein, 1967) of the autonomic and subjective report responses to the independent variables.

Turning to the autonomic-behavioral relationship, there are fewer relevant studies, and the behaviors measured are more heterogeneous than the subjective report measures above. Frazier, Weil-Malherbe, and Lipscomb (1969), Harleston et al. (1965), Jones and Johnson (1973), and Mandler, Mandler, Kremen, and Sholiton (1961) found significant autonomic-behavioral correlations; but Frazier et al. and Jones and Johnson found a greater number of nonsignificant correlations, and Mandler and Kremen (1958) found no significant correlations. In addition, the Harleston et al. and Mandler et al. studies showed performance decrements with increasing autonomic arousal; whereas Frazier et al. found improvement and Jones and Johnson generally found greater

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[1] Supplementary analysis on data collected for E. Proctor and M. Kinsbourne study entitled "Lateral Eye Movements and State Anxiety," submitted for publication, 1973.

willingness to enter a risky situation rather than avoidance. In fact, Jones and Johnson concluded that, in conditions in which the consequences were imminent, arousal seemed to be the result of making a risky choice rather than the cause. Similarly, Harleston et al. suggested that differences in arousal may result from success or failure on a task. Rappaport (1971) found no significant autonomic differences between high- and low-avoidance subjects. Lanzetta and Kleck (1970) reported a significant correlation indicating that those subjects who produced the greatest autonomic response exhibited the least observable response to stress as judged by observers. Other incongruities were observed by Leitenberg, Agras, Butz, and Winze (1971) who reported several phobia therapy cases in which behavioral improvement was accompanied by increased or unchanged autonomic arousal. Likewise, Jones and Johnson reported experiments in which the behavioral measure was affected significantly by the independent variable but the autonomic measures were not.

The inconsistencies within and between the studies reviewed above and the insubstantiality of even the significant correlations have often been attributed to certain annoying characteristics of autonomic responses rather than to inherent weakness in the autonomic-subjective and autonomic-behavioral relationships. Lacey (1967) has pointed out that autonomic responses exhibit very low or no intercorrelations, are produced in reliably different patterns by different situations, and are subject to systematic individual differences in patterns of response.

However, a growing number of psychologists would agree with Weinstein et al. (1968) who argued that some discrepancies among autonomic, subjective, and behavioral response measures would remain even if the assessment of each was methodologically adequate. Mordkoff (1964) tested the autonomic-subjective relationship under some of the most favorable conditions (semi-continuous subjective report while under stress, multiple autonomic variables recorded continuously, and intra-individual analysis) and found a mean multiple correlation of 0.508. Although many of the other studies reviewed also included several autonomic variables or incorporated other methodological refinements, this was the highest autonomic-subjective correlation found; and a correlation of 0.5 accounts for only 25% of the variance. In other words, by determining whether sympathetic-like activity increases after introduction of threat in such studies, one can predict that self-report of anxiety will increase or decrease, respectively, but not how much--changes in the two variables fall short of temporal synchronization or correspondence in magnitude. As for autonomic-behavioral relationships, the situation is worse because not even the direction of behavioral change can be predicted. No generalizations can be made except that subjects who show qualitative or quantitative differences on a behavioral response often differ in their autonomic response to threat as well.

In general, autonomic-subjective and autonomic-behavioral relationships are so weak and inconsistent that subjective report of anxiety and

overt behavior under stress could not depend completely on the activity of the autonomic nervous system. Autonomic arousal is not a sufficient cause. Taken together these studies argue against a direct causal or controlling relationship between autonomic and subjective or behavioral responses. Instead, in most of the studies cited, there is evidence that these are three different types of responses, which diverge in their functions and in their control by external contingencies; they may be loosely related simply because they are responses to the same situation with the same stimulus conditions and same behavioral requirements.

The main contention of this paper is that autonomic variables do not play a direct controlling role in these relationships, but that they can play an indirect or mediated controlling role without being the sole controlling factor. Under what conditions can autonomic variables act as stimuli to subjective report of anxiety and behavioral reactions under stress rather than simply as one of the organism's responses to stress? Autonomic changes often are not perceived and are rarely perceived very accurately; but when the individual is aware of autonomic changes, this imperfect awareness (rather than the actual autonomic changes) is one controlling factor for subjective report of anxiety and behavior under stress. Thus, the cognitive mediation (the imperfect awareness) is more important than the actual autonomic activity. The other important controlling factor is also cognitive. This position differs from James' theory and Wolpe's assumptions in the contention that there is not a one-to-one relationship

between actual autonomic arousal and its perception and in the addition of a second determining condition.

## Awareness of Autonomic Arousal as One Controlling Factor

### Condition of degree of arousal

Awareness of autonomic arousal normally arises from the individual's perception of his own internal responses. Cannon (1927) argues that "the viscera are relatively insensitive structures (p. 111)" in refuting James' claim that different subjective emotions depend directly on the perception of different patterns of visceral response. However, the existence of afferent nerve fibers from the viscera to the central nervous system is well established (Fehr & Stern, 1970; Lacey, 1967). Furthermore, we are proceeding in a different direction than James' hypotheses that visceral responses are the only (sufficient) cause of subjective emotion and that every subjective emotion is produced by a different pattern of visceral responses, or the comparable hypotheses made by Wenger for emotional behavior. A demonstration that there is some perception of autonomic responses, no matter how vague, is adequate for the present contention that awareness of autonomic arousal contributes but is not sufficient. In fact, poor perception would help to explain the weak relationships between actual autonomic measures and subjective report or behavioral measures in the studies reviewed above.

It does not seem to be specific and subtle changes, such as

measured in most of these studies, which are perceived. At least they are not accurately identified and quantified in stressful situations. Strahan (1971) obtained a significant correlation between reported perception of heart rate changes and actual changes, but no significant relationship for finger temperature. The correlation between reported and actual palmar sweating found by Pillard et al. (1966) was significant only when one-tailed tests were used. A series of studies conducted by Mandler and his associates (Mandler & Kremen, 1958; Mandler et al., 1961; Mandler, Mandler, & Uviller, 1958) generally indicated very weak relationships between perception of autonomic activity and actual activity. As a group, high perceivers overestimated their autonomic reaction whereas low perceivers underestimated. Mandler and Kremen entertain the possibility that autonomic report depends only slightly on the actual occurrence of autonomic activity. Proctor and Kinsbourne[1] found a nonsignificant negative rank-order correlation between pulse rate elevation and reported cardiac sensations. Like Mandler and his colleagues, they found that high perceivers overestimated and low perceivers underestimated. A weak relationship is also indicated by research on operant conditioning or voluntary control of autonomic responses. In most studies, significant control of specific autonomic variables has been obtained only when extrinsic feedback is provided; this has been attributed to the

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[1] E. Proctor and M. Kinsbourne, study entitled "Lateral Eye Movements and State Anxiety," submitted for publication, 1973.

insufficiency of the normal feedback for indicating to the subject whether he has performed the correct response (Brener & Hothersall, 1966). In fact, our autonomic perception is so poor that subjects who are misinformed concerning the correct autonomic response may learn it with extrinsic feedback, in spite of their belief that they are changing a different bodily process (Ascough & Sipprelle, 1968; Headrick, Feather, & Wells, 1971).

One possibility is that the individual is aware of summated or extreme autonomic activity but cannot discriminate slight changes in specific autonomic variables, such as minor changes in heart rate or respiration, or especially less obtrusive changes such as vasomotor and blood pressure responses or skin resistance. Mandler (1962) makes just this point in his discussion of the importance of a physiological variable being discriminable if it is to be a controlling, or "psychologically functional," physiological variable. When autonomic changes are very slight, as were some of the significant autonomic differences in the review earlier in this chapter, the individual probably is not aware that there has been a change. Even with greater changes in arousal, the individual may only be aware that *something* feels different; if we ask him whether it was his heart rate, stroke volume, respiration, perspiration, blood pressure, or peripheral blood volume, or to estimate the size of the change, we may find him unable to make these distinctions except for sudden or gross changes in some of the more noticeable variables. A list of the reactions

which are most likely to be perceived in everyday life has been compiled by Stern and Higgins (1969). Their subjects most often reported the following as occurring prior to an important event: nervous stomach, sweaty palms, general restlessness, increased heart rate, and awareness of heart beat.

There is evidence that diffuse and global changes in autonomic activity are perceived, at least as a change in arousal. Most of this evidence comes from studies involving administration of epinephrine, a sympathomimetic drug. The adrenal medulla, which secretes epinephrine, may be considered to be a part of the sympathetic nervous system (Turner, 1966), and administration of epinephrine is one way to vary the level of sympathetic activity experimentally. Some relevant effects follow, as summarized from several sources (Best & Taylor, 1966; Goodman & Gilman, 1970; Turner, 1966). Epinephrine is a cardiac stimulant, and its vasodilator effects dominate the circulatory pattern in most cases. It increases the force and amplitude as well as the frequency of contraction of the heart. When administered or absorbed slowly, increased cardiac output generally results in a moderate increase in systolic blood pressure, but diastolic pressure usually falls. Circulatory effects depend on the dosage; small doses decrease peripheral resistance due to activation of the beta receptors and consequent vasodilation in the skeletal muscles, but larger doses also act on the alpha receptors which produce vasoconstriction in the skin, mucosa, and kidneys. Epinephrine also

stimulates respiration, increasing respiratory rate and tidal volume. In addition, epinephrine stimulates skeletal muscle, one prominent effect being muscle tremor.[1] Epinephrine does have central nervous effects but is not considered to be a powerful central nervous system stimulant.

Many studies support the conclusion that subjects perceive, in at least some general way, the pharmacological effects of epinephrine. Some of the earliest studies (Cantril & Hunt, 1932; Landis & Hunt, 1932; Marañon, 1924) giving this indication lacked adequate controls and standardized measures of perception, but later studies have supported their findings. Schachter (1966; Schachter & Singer, 1962) found that epinephrine subjects reported greater palpitations and muscle tremor than placebo subjects with the same expectations. Hawkins, Monroe, Sandifer, and Vernon (1960) used a continuous infusion procedure with several subjects who were not informed as to whether epinephrine, saline, or norepinephrine was being administered; infusion of epinephrine was always detected immediately. Frankenhaeuser and Järpe (1963) infused several dose levels of epinephrine and a placebo; although their reporting of the data is inadequate, the study is interesting because of the low dose levels investigated (between 0.05 micrograms/kilogram/minute and 0.20

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[1] Although this effect might seem to have little to do with autonomic arousal, recent evidence (Bowman & Nott, 1971; Marsden, Foley, Owen, & McAllister, 1967; Marsden, Gimlette, McAllister, Owen, & Miller, 1968; Marsden & Meadows, 1968; Marsden, Meadows, Lange, & Watson, 1967) indicates that this effect is mediated directly by the peripheral beta-adrenergic receptors, although the exact mechanism is uncertain.

micrograms/kilogram/minute. Subjects noted palpitations most frequently and then dry mouth, numb limbs, pounding in head, chest, or abdomen, feelings of unreality and/or distance from surroundings, headache, and nausea, in that order. Presumably, the muscle tremor which was observed by the experimenters at even the low dosages was also noted by the subjects. The frequency of the symptoms was greatest at the highest dosage and was practically absent with the placebo. The authors report that frequency of reported symptoms did not correspond to dose level in the intermediate conditions; this is what we would expect from organisms that can discriminate the gross differences in diffuse autonomic arousal but are incapable of distinguishing slight gradations.

Since perception is not perfect, it alters the relationship between autonomic arousal and subjective report or behavior; the evidence points to a controlling role for perception, or more generally awareness, rather than for actual autonomic arousal. Pillard et al. (1961) and Proctor[1] found stronger relationships between "perceived" autonomic activity and self-report of anxiety than between actual autonomic activity and self-report. Similarly, Mandler and Kremen (1958) found the relationship with performance under stress to be stronger for "perceived" than for actual autonomic activity.

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[1] Supplementary analysis on data collected for E. Proctor and M. Kinsbourne, study entitled "Lateral Eye Movements and State Anxiety," submitted for publication, 1973.

Although perception of one's own autonomic activity is the normal method of obtaining information concerning the level of arousal, awareness of autonomic arousal can also be obtained from observation of a measuring device or information from other individuals. As with perception, these methods can be inaccurate; and the inaccuracies provide a test of whether it is the actual arousal or the awareness, that is, the cognition of degree of arousal, that plays the controlling role in subjective report of anxiety or maladaptive behavior under stress.

Valins and Ray (1967) have suggested that even in the desensitization of phobias it may not be necessary that relaxation actually inhibit autonomic arousal but just that the patient believes that he is less aroused in the presence of the phobic stimulus. As a test of this hypothesis, they dissociated the cognition from the actual arousal and manipulated the cognition as the independent variable by providing subjects with arbitrary heart-rate feedback. Subjects who were induced to believe that their heart rates increased in anticipation of electric shocks but not when viewing slides of snakes or a live snake showed greater approach behavior toward a live boa constrictor than control subjects. These differences were significant for those subjects who previously had never touched a snake (viewing slides) or who previously reported extreme fear of snakes (viewing live snake). Both experimental and control groups reported less subjective fear at the end than at the beginning of the experiment. The authors concluded that cognitions about bodily changes may be more

important than whether these cognitions are veridical in producing change in behavior. This conclusion is consistent with an earlier study (Valins, 1966) in which manipulation of bogus heart-rate feedback affected male subjects' preferences for pictures of seminude females. Although usually related to actual arousal, albeit imperfectly, the cognition of arousal can have powerful effects even when highly inaccurate.

A summary of natural cases and research in which autonomic arousal has been perceptibly altered was delayed in order to complete the argument for the importance of perceived or otherwise cognized arousal, as opposed to actual arousal, as a controlling factor. The following studies do not deal with the discrepancies between actual arousal and cognized arousal, but they do examine the controlling nature of cognized arousal for subjective report or behavior. In the following studies, the cognition is based on perception of gross differences in autonomic arousal.

The contribution of awareness of autonomic arousal has been investigated in cases in which accidents or disease have deprived individuals of afferent impulses from autonomically innervated organs. In support of his theory, James (1894) cited one case in which an almost complete reduction in subjective emotion was reported by a patient who suffered a lack of visceral, cutaneous, and muscular sensations; similarly, two subjects with hypnotically induced anesthesia of the viscera reported no subjective emotion following suggestions which otherwise aroused subjective report of emotion. These cases were originally reported by Sollier (1894).

James (1884, 1890, 1894) discussed other cases which are ambiguous on the question of subjective emotion but in which visceral anesthesia was accompanied by no obvious reduction in emotional behavior. Dana (1921) reported no change in emotional feeling in a patient with a cervical lesion (incomplete) of the spinal cord, but he does not specify whether this conclusion was reached from observations or subjective report.

Hohmann (1966) conducted a more systematic investigation of the effects of lesions at different levels of the spinal cord on reported emotional feelings in 25 paraplegics. Hohmann believes that his being a paraplegic himself overcame his subjects' tendency to attempt to appear "normal." Except for subjects with high cervical lesions, transections of the cord were judged to be clinically complete on the basis of neurological examination. Subjects reported diminishment in subjective fear or anxiety (and in all other emotions tested, except sentimentality) as compared to instances prior to injury. Subjects stated that it was the loss of perceptions of physiological disturbances which made their subjective fear less. Hohmann's report concentrates on the fact that reported emotional feeling was reduced in these men; however, it appears that even those subjects who had no perception of autonomic arousal from below the neck talked in terms of having some feeling of subjective fear, indicating that in these subjects, for whom autonomic arousal can no longer function as a cue, awareness of autonomic arousal is not necessary for subjective report of some level of anxiety, although it seems to be necessary for

what the subjects might label as the complete experience of anxiety. Hohmann found the decrease in reported subjective emotions to be proportional to the amount of disruption of the autonomic nervous system sensation (that is, the more cephalad the lesion, the greater the decrease). However, the paraplegics reported their ability to act appropriately in situations in which they would have felt fear prior to their injuries, such as avoiding a realistic danger. No information was presented on whether these subjects ever behaved maladaptively or exhibited inappropriate avoidance or behavioral decrements under stress. Conflicting data on subjective report have been reported by Hester (1971), who found that paraplegic subjects with cervical cord transections did not differ significantly from normals in the subjective anxiety reported.

Autonomic arousal has also been inhibited pharmacologically in order to study its relationship to reported subjective anxiety. Although some of this work seems based on the view that subjective anxiety is directly dependent on autonomic activity, this view has not been borne out by the results. Propranolol blocks access to the beta-adrenergic receptors of the sympathetic nervous system, thereby inhibiting increases in heart rate and contractile force and muscle tremor which would otherwise be produced by sympatho-adrenal activity (Goodman & Gilman, 1970). The authors cited here believed that the doses used in their studies were too small to produce central nervous system effects. After finding that propranolol reduced tachycardia in anxious patients (Turner, Granville-

Grossman, & Smart, 1965), Granville-Grossman and Turner (1966) investigated the effect of propranolol on subjective anxiety in characteristically anxious outpatients. Although a psychiatrist (blind to the drug condition) rated significantly more subjects as less anxious after propranolol than after a placebo, subjects rated themselves as significantly more improved after propranolol than after a placebo only for autonomic symptoms (palpitations, sweating, diarrhea, etc.). Subjects did not rate themselves as feeling significantly greater improvement after propranolol than after the placebo for all symptoms, for all mental symptoms, or for all nonautonomic bodily symptoms. Although no data are presented, Holmberg, Levi, Mathé, Rosén, and Scott (1967) report that, in their labile hypertensive and alcoholic patients, subjective "emotional reactions are slightly reduced at repetition of the stressor procedures following adrenergic beta-receptor blockade (p. 209)." A study by Cleghorn, Peterfly, Pinter, and Pattee (1970), using normal subjects under transitory stress, is more relevant. Mildly stressful situations were suggested to the subjects under hypnosis; scores for verbal report of anxiety were not significantly different following administration of propranolol or a placebo. This was possibly due to the fact that none of the subjects reported any palpitations or other such physical symptoms even during the placebo trials. On the basis of their research and other work with propranolol, the authors concluded that autonomic arousal can contribute to report of subjective anxiety and that reduction in the subjective anxiety reported

following propranolol administration is due to a reduction in bodily sensations. They believe this to be a cognitive rather than a subliminal biological effect; specifically, the beta receptors contribute to subjective report of anxiety only "when their activation is associated with physical sensation which some persons learn to interpret as a signal of danger (p. 271)."

Other evidence concerning the contribution of awareness of autonomic arousal comes from Schachter's work (1966; Schachter & Singer, 1962). One of Schachter's hypotheses is that "the individual will react emotionally or describe his feelings as emotions only to the extent that he experiences a state of physiological arousal (1966, p. 197)." Arousal is thought to motivate the individual to understand and label his state. This hypothesis received some support from an experiment in which subjects received an injection of epinephrine or a placebo and then were exposed to a stooge who acted euphoric or angry. Although this study did not use a threatening situation, Schachter maintains that the same processes apply. Both self-report and behavioral measures were taken. Placebo subjects scored as significantly less euphoric or angry than epinephrine subjects given the same additional manipulations only on the behavioral observation in the anger condition. Schachter explains this as partly due to endogenously produced arousal in some placebo subjects. When placebo subjects whose heart rate increased or stayed the same were compared to placebo subjects whose pulse rate decreased over the course of

the experiment (presumably due to habituation), the differences were significant for both anger and euphoria behavioral measures. Likewise, there were significant differences for the behavioral measures when certain "self-informing" epinephrine subjects who were not deceived by the manipulations were excluded from the analysis. Schachter concludes that the experience of arousal is a necessary condition for report of subjective emotions, including anxiety, and for the behavioral reaction, and that, in appropriate situations, the intensity of these responses will reflect the degree of arousal. Although Schachter implies that this experience of arousal depends directly on actual arousal, we have already noted in discussing Valins' work (1966; Valins & Ray, 1967) that manipulation of the cognitive awareness of arousal can have the same effect and might override actual arousal.

### Attribution of source of arousal

Another aspect of Schachter's work has taken research on reported subjective anxiety and overt behavior under stress in a novel, although obvious, direction. Schachter's main hypothesis is that a general pattern of sympathetic discharge is characteristic of all emotional states, but that this discharge is labeled and interpreted in terms of the precipitating situation; in other words, the cognitive attribution exerts a steering function. Schachter and Singer's study demonstrated that subjects who received epinephrine injections would label their subjective state differently

depending on the circumstances to which they attributed their arousal. Subjects who were not aware that the epinephrine injection produced their arousal presumably thought their arousal was an endogenous reaction to their interaction with the stooge--and they described themselves as happy or angry (actually less happy on the scale) and acted euphoric or angry, respectively, depending on the stimulus situation (euphoric or angry stooge). Subjects who were aware that their arousal was produced by the injection were significantly less affected, on both the subjective report and behavioral measures, by the stooge manipulation. Since the actual arousal was presumed to be the same in all groups which received epinephrine, Schachter interprets this as a cognitive factor and concludes that, in addition to the awareness of arousal, a second necessary condition for the report of a particular subjective emotion is an appropriate attribution of the source of the arousal.

The pharmacological effects of epinephrine closely approximate the physiological reactions which can often be measured when a subject is under stress or reports he is experiencing subjective anxiety (Ax, 1953; Breggin, 1964; Funkenstein, 1955; Martin, 1961). In addition, many of these reactions can be perceived, as discussed earlier. However, the data by and large agree with Schachter's emphasis on the necessity of an appropriate causal attribution for the arousal before one will label himself as subjectively anxious. In an extensive review of epinephrine-administration studies, Breggin (1964) concludes that subjects have only

reported subjective anxiety if the experimental situation was inherently threatening or if they were vividly reminded of other threatening situations. Most subjects have simply reported their physiological sensations or have reported feeling "as if" emotional, recognizing the similarity of their physiological symptoms to those aroused in threatening situations; they presumably did not label themselves as subjectively anxious since their arousal had apparently been caused by a drug rather than any threat.

An individual labels his state of cognized arousal in terms of certain cognitive and situational factors (and then acts accordingly) when he does not have a more salient alternative explanation. Normally, the cognitive or situational factors produce the arousal and the causal attribution is obvious. However, Schachter showed that it is possible to manipulate the attribution of drug-induced arousal in an ambiguous situation and that this manipulation changes behavior and the label on the subjective feeling. Several important studies have shown that the attribution of naturally occurring autonomic arousal is just as manipulable, and this dissociation has demonstrated that the attribution is indeed a controlling factor.

Ross, Rodin, and Zimbardo (1969) introduced threat of shock in a study purporting to deal with the effects of noise on performance. The experimental group, or noise-attribution subjects, were told that loud noise often produces side effects such as tremor, palpitations, visceral upset, and increased respiration rate (symptoms actually common when anticipating shock); thus, these subjects were predisposed to attribute

their shock-produced arousal to the noise. The control group of shock-attribution subjects was informed that dizziness, headache, ringing in the ears, and weariness were possible side effects of noise and thus had no alternative explanation for shock-anticipation arousal. Subjects were given two puzzles to solve in a limited time period. The puzzles were actually insoluble, but subjects were told that solution of one would avoid the shock and that solution of the other would win a monetary reward. Noise-attribution subjects spent significantly less time working on the shock-avoidance puzzle than shock-attribution subjects. In addition, the number of people spending more than half of their time on the shock-avoidance puzzle was significantly less for the noise-attribution group. On an open-ended questionnaire at the end of the experiment, only 10% of the noise-attribution subjects reported fear of shock, but 75% of the shock-attribution subjects reported fear or physiological symptoms specifically attributed to shock-anticipation. Thus, misattribution of the arousal produced by anticipation of shock to an extrinsic source had beneficial effects, and the authors conclude that a technique which makes people feel and act less afraid has therapeutic potential.

Similarly, through re-attribution of arousal to an extrinsic source (a pill which was actually a placebo), Nisbett and Schachter (1966) demonstrated a decrease in perceived pain and a willingness to tolerate higher intensities. Storms and Nisbett (1970) obtained a reduction in insomnia in subjects who were convinced that their wakefulness was due to an

extrinsic source (a placebo pill). Thus, the important inference seems not to be merely "I am (or am not) aroused" but "That stimulus has (or has not) aroused me."

## Implications and Critique

The research reviewed in this introduction presents some promising alternatives for reducing subjective distress or maladaptive behavior in threatening situations. We have reviewed the evidence concerning three independent variables: actual autonomic arousal, awareness or cognition of the degree of autonomic arousal, and causal attribution of the cognized arousal. Normally, the effects of autonomic arousal seem to be mediated first by perception, or more generally, cognition, of arousal and secondly by the cognitive attribution of any cognized autonomic arousal to an emotional or nonemotional source. From the research reviewed, cognitive factors apparently are of major importance. Our perception of autonomic arousal is so poor that in some cases the cognition of arousal may be independent of actual arousal. Although normally arising from perception of autonomic arousal, the cognition of arousal rather than actual arousal seems to be a controlling variable for the subjective report of anxiety or avoidance behavior, but the cognition of arousal is not sufficient--the cognitive attribution of arousal to an appropriate source is also necessary.

Thus, we have arrived at an intuitively pleasing synthesis of much

of the preceding research, a synthesis made possible and interesting by the work of Schachter (1966; Schachter & Singer, 1962), Valins (1966; Valins & Ray, 1967), and Ross et al. (1969). However, we actually have a set of hypotheses or mini-theories which have not yet been well substantiated. Further research is necessary for the following reasons:

1. The Schachter and Singer study did not furnish strong evidence for the hypothesis that, given an appropriate attribution, subjects would report emotion or act emotional if they perceive physiological arousal. Although epinephrine subjects had significantly higher heart rates and reported significantly more tremor and palpitations than placebo subjects, no group given epinephrine (regardless of the attribution induced) differed significantly from placebo subjects except on the anger behavioral measure. This was true even though subjects who showed no arousal on heart rate were excluded from the analysis. Significant results on both the anger and the euphoria behavioral measures were produced only when (a) "self-informing" subjects in the groups which had not been given the drug attribution were excluded from the comparison with placebo subjects, or (b) placebo subjects who did not show arousal on pulse rate were compared to those "self-arousing" subjects who did. Comparable data for the subjective report measures are not reported in either case; in addition, the anger behavioral measure was already significant without these internal analyses. The differences within the euphoria placebo group are questionnable because pulse rate was measured after the behavioral measure, for

which a higher score represented greater movement.

Other studies are cited by Schachter to bolster his case. Two studies (Latané & Schachter, 1962; Singer, 1963) used rats to prevent self-informing tendencies, but these subjects are not appropriate for a theory which proposes that physiological arousal is controlling because it produces a need to evaluate and label one's state. A third study (Schachter & Wheeler, 1962) included a chlorpromazine group to widen the range of autonomic arousal. Epinephrine subjects showed more emotional behavior than placebo subjects; both of these groups showed more emotional behavior than chlorpromazine subjects--which is not surprising since chlorpromazine is a powerful central nervous system sedative (Goodman & Gilman, 1970). These studies relate only to emotional behavior and are not entirely convincing on that point.

2. Although hypotheses and conclusions are often stated in terms of both subjective labeling and overt behavior, it is not evident that these two variables respond to actual arousal, cognition of arousal, or attribution of arousal manipulations in the same way. Although he has not always made a distinction between subjective emotion and emotional behavior, Schachter (1966) concluded at one point that, in the absence of visceral impulses, a person could act emotionally but could not feel subjective emotion. Clearly, this was not evidenced in his own work. Like Schachter, Valins and Ray (1967) found evidence for the dependence of emotional behavior, in this case avoidance, but not subjective report on

the degree of cognized arousal. On the other hand, Hohmann (1966) found that paraplegics reported a greater reduction in subjective emotion after injury than in emotional behavior.

3. Two recent studies failed to replicate the effects for cognition of degree of arousal reported by Valins and Ray (1967). Rosen, Rosen, and Reid (1972) did find that subjects who reported high fear of snakes tended to agree that "When I see a snake my heart rate increases." Subjects who met both of these criteria and who could not touch a snake heard their bogus heart rate increase during snake slides or shock slides. Although their ratings of nervousness during the slides were in accord with their bogus heart rate, neither an avoidance posttest nor any of three subjective report measures of fear of snakes differed significantly for the two groups. The authors conclude that subjects whose bogus heart rate remained constant during snake slides devalued the fearfulness of the slides rather than re-evaluating their fear of live snakes. Although the results do not replicate the Valins and Ray (1967) study, a cognitive re-evaluation did occur. In a similar study (Suskinsky & Bootzin, 1970), no significant differences were found between pretest and posttest on behavioral avoidance, and experimental subjects did not differ significantly from the control groups on either the behavioral posttest or on self-ratings of disturbance or discomfort during the slides.

4. The Valins (1966) and Valins and Ray (1967) studies leave open the possibility that their effects were mediated by differences in actual

arousal. No measures of actual heart rate were taken in either study, but Valins (1966) stated that experimental subjects (who were induced to believe that their heart rate changed during certain slides) reported significantly fewer palpitations than did control subjects. Without presenting any details, Valins (1966) also mentioned that this difference had been replicated and that the replication also indicated that experimental and control subjects did not differ on galvanic skin response and heart-rate measures. Valins concluded that the effects of bogus heart rate were not physiological and that bogus feedback actually diverted attention from true arousal.

Some doubt is cast on this conclusion by other research which has included physiological measures as an integral part of the design. Goldstein, Fink, and Mettee (1972) performed two experiments. In the first, male subjects viewed slides of nude females; bogus heart rate increases were accompanied by greater ratings of attractiveness and by significant deviations (increases or decreases) in actual heart rate, but absolute deviations in heart rate were not significantly related to reported attraction. In the second study, males viewed male nudes; speedups in bogus heart rate did not significantly affect actual heart rate or ratings of offensiveness, but absolute deviations in heart rate were significantly related to report of offensiveness. The authors concluded that physiological arousal also acts unconsciously rather than merely as a cognitive cue and that it is the determining factor when actual arousal and bogus

cognitions are contradictory, as may be the case in highly emotional situations.[1]

Stern, Botto, and Herrick (1972) also replicated the effect of bogus feedback on ratings of attractiveness described by Valins (1966) and presented evidence for actual autonomic differences which could account for such results. All of the studies on bogus feedback reported in this paper have used auditory stimuli which were described to subjects as being the sound of their own heart beat, picked up by a microphone on their chest, and unavoidably amplified to an audible level. For control subjects the same sounds were described as "extraneous sounds." Stern et al. found effects similar to those in the Valins study for rated attractiveness of slides of seminude females, and they found significant differences between experimental and extraneous sound subjects on actual heart rate.[2] When extraneous sound subjects were told to attend to the sounds in a second experiment, their subjective ratings and heart-rate responses were very similar to those given by bogus heart-rate subjects. These results held for a third experiment using slides of dead or injured people and with within-subject analyses. The authors' explanation based on

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[1] Alternatively, subjects in the second study may not have accepted the bogus feedback and may have used their actual autonomic changes as a cognitive cue; or, more likely since heart-rate deviations were small, actual autonomic changes may have been the result of, rather than the controlling factor for, the subjects' being shocked or offended.

[2] Although averaged over constant feedback and change trials together for each group.

differences in attention does not obviate the need for a cognitive reevaluation as the next step. However, like Goldstein et al. (1972) they have demonstrated autonomic results of the manipulation, and they have also suggested a controlling condition for that arousal.

5. Autonomic measures in past studies have not been adequate to substantiate Schachter's hypothesis that, as long as some arousal is present, extrinsic (nonemotional) and intrinsic (emotional) attributions have different effects even when arousal is identical. Schachter and Singer (1962) and Ross et al. (1969) found that attribution of arousal to an extrinsic source, drug or noise, respectively, reduced the report of subjective emotion and emotional behavior. Ross et al. did not include any measures of actual arousal. Schachter and Singer did not test for differences between the attribution groups on their autonomic measure, pulse rate. However, pulse rate was measured so late in this study (after interaction with the stooge) that it probably would not have reflected accurately any earlier differences in arousal which could have influenced the results.

6. Attribution has not always been found to be an effective manipulation in threatening situations. Nisbett and Schachter (1966) were interested in the effects of attributions of arousal on reported sensitivity to pain and pain tolerance. Although they found the predicted effects on these measures, the study also allows an appraisal of the effects on reported fear of shock and worry. Self-report data were gathered after the attribution of arousal to an extrinsic source (placebo) or intrinsic source (shock

anticipation) was induced, but before shocks began. Reported fear and worry about the effects of shock apparently did not differ in relation to the attribution condition according to the data presented (p. 232).

Another interesting finding in this study is that subjects who were induced to expect shock to be very aversive did not accept the extrinsic (placebo) attribution to the same extent as subjects who were led to expect mild shock. The authors suggested that there are limits to the plausibility of attributions. A similar point is indicated by the Rosen et al. (1970) study in which some subjects who had previously reported extreme fear of snakes devalued the stimulus slide rather than attributing their lack of arousal (bogus) to snakes. Schachter and Singer also had some difficulty in inducing a misattribution in some intrinsic attribution subjects; these self-informing subjects correctly attributed their arousal to the injection.

7. The effects of perceptible actual arousal, induced cognition of arousal, and attribution of arousal on subjective report and behavior have not been assessed in the same experiment. Experiments manipulating attribution of arousal logically require arousal; however, most such studies since Schachter and Singer's have used naturally occurring autonomic arousal and have not examined the effects of differences in arousal. Studies using bogus feedback to induce cognitions of arousal make use of appropriate attributions but usually deal only with intrinsic attributions.

## Statement of the Problem

Since actual autonomic arousal, cognition of arousal, and attribution are interrelated in everyday life, all three were manipulated in this investigation. They were manipulated and their effects measured in such a way as to clarify some points left unsettled by previous studies.

Since the utility of the three independent variables for reducing reported subjective anxiety or maladaptive behavior under stress hinges on their effects in real-life situations, rather than in comparatively safe and ambiguous laboratory settings, a relatively common stressful situation was used. This project used subjects who were awaiting oral surgery. Dental treatment was chosen because it is needed by most people, and it generates reported subjective anxiety (Dabbs, Douglass, & Strachan, 1971; Pillard & Fisher, 1970) and oftentimes maladaptive avoidance behavior, ranging from people who procrastinate to people who will approach a dentist only when provoked by severe pain. The Schachter and Singer, Valins and Ray, and Ross et al. studies used college students (a notoriously introspective group) in contrived experimental settings; findings from the present study could have direct applications to the dental situation, at least, and theoretical relevance to other real-life fears and phobias.

Despite its differences from previous studies, several hypotheses derived from those studies were advanced in the present investigation to focus attention on the main questions of interest. Before presenting these

hypotheses formally, let us consider what the important questions would be from the dentist's point of view. Many dental patients anticipate pain or discomfort and presumably exhibit some degree of autonomic arousal. In addition, the dentist may administer a local anesthetic containing epinephrine, thereby increasing arousal. If a dentist is interested in reducing the subjective anxiety of his patients and in preventing such maladaptive behavior as avoidance or procrastination of dental treatment in the future, what should he do? Should he use the anesthetic preparation containing epinephrine as seldom as possible or does the increase in arousal have no greater subjective or behavioral effects than would be experienced without the epinephrine? If the epinephrine increases subjective report of anxiety and behavioral disturbances, can these effects be prevented if the patient is informed that certain physiological symptoms are to be expected and are due to the drug injected? Alternatively, will providing the patient with the cognition that his arousal is less than or equal to the norm or his own baseline reduce the effects of epinephrine-induced arousal? Will such cognitions change the patient's actual arousal? Will similar procedures reduce the subjective anxiety reported and the avoidance tendencies of patients who receive an anesthetic without epinephrine? Will the plausibility of the latter cognitive manipulations be limited by the level of actual arousal? Do these cognitive manipulations have unfavorable effects on subjective report of anxiety or behavior by directing the patient's attention to his arousal?

The independent variables were Actual Arousal, Cognition of Arousal, and Attribution of Arousal. The intent was to compare perceptible differences in Actual Arousal so a drug manipulation was used; the two conditions were Epinephrine and No Epinephrine. To induce Cognition of Arousal, information was given to the subject once rather than continuously in order to eliminate differential external attention and to prevent the manipulation from detracting from actual autonomic activity. Subjects were simply told that they were Aroused or Unaroused or given No Information. Attribution was also manipulated as simply as possible; there was an Intrinsic condition and an Extrinsic condition in which subjects were told that the drug caused arousal. A factorial design was used so that interactions between the independent variables could be assessed. Autonomic measures (pulse rate and blood pressure) were included as manipulation checks, and Post-Manipulation measures were dependent variables. Other dependent measures were Subjective Report and two behavioral measures, Tendency to Avoid and Observed Behavior (nervous or anxious overt behavior).

The main hypotheses follow. The first three are presented for Subjective Report, Tendency to Avoid, and Observed Behavior since the exact points of divergence of the subjective and behavioral measures, although probable, could not be predicted from the available literature. Of course, the data were examined in additional ways to answer the questions posed above; specific predictions could not be made on all points from

previous data.

The hypotheses were:

1. Epinephrine subjects would have higher scores on Subjective Report, Tendency to Avoid, and Observed Behavior than No Epinephrine subjects, but only in the Intrinsic Attribution condition.

2. Aroused subjects would have higher scores on Subjective Report, Tendency to Avoid, and Observed Behavior than No Information subjects, but only in the Intrinsic Attribution condition. Unaroused subjects would have lower scores on Subjective Report, Tendency to Avoid, and Observed Behavior than No Information subjects, but only in the Intrinsic Attribution condition.

3. The Intrinsic Attribution subjects would have higher scores on Subjective Report, Tendency to Avoid, and Observed Behavior than the Extrinsic Attribution subjects, but only in the Epinephrine or Aroused conditions.

4. Cognition of Arousal conditions would not differ on Post-Manipulation Pulse Rate, Post-Manipulation Systolic Blood Pressure, or Post-Manipulation Diastolic Blood Pressure.

# METHOD

This study was conducted at the Oral Surgery Clinic of the University of North Carolina School of Dentistry. This clinic has six individual operatories in which teeth were extracted by a senior dental student, assisted by a student dental assistant or another dental student, for a nominal fee. Only a local anesthetic was used for these extractions.

## Subjects

The subjects were 96 patients attending the Oral Surgery Clinic. Because of a shortage of patients, both sexes (48 of each) were used. Only patients who were between the ages of 18 and 50, inclusive, and whose blood pressure did not exceed 140 millimeters of mercury systolic or 100 millimeters of mercury diastolic were asked to participate. In addition, the sample was confined to patients who were having extractions of only mandibular teeth because the anesthetic injection is approximately the same regardless of the tooth (or teeth) to be extracted; this is not true of maxillary extractions. The locations injected for maxillary extractions vary in pain sensitivity and consequently produce different autonomic reactions (Vernale, 1960). Thus, use of only mandibular cases kept the injection site as constant as possible and kept injection pain,

which otherwise might have affected the subject's psychological and physiological responses, fairly constant as well.

## Overview

After the subject was seated in the dental chair, baseline pulse and blood pressure measures were taken, as is routine in the Oral Surgery Clinic. Next the study was introduced and the patient's consent obtained. The anesthetic was then administered by the dental student, following which he and his assistant left the room.

Approximately one minute after completion of the anesthetic injection, pulse and blood pressure were measured again. At this point the manipulations of Cognition of Arousal and Attribution were introduced. Pulse and blood pressure were then taken a third time. Subsequently, a questionnaire was administered concerning self-report of subjective anxiety, tendency to avoid, perception of actual arousal, and attribution of arousal.[1] All subjects were given preliminary instructions orally, and instructions and items within the questionnaire were read to the subject if

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[1] Another measure, subjective anxiety prior to the injection and other manipulations, was originally included in the questionnaire as a measure of initial differences between the groups of subjects. This measure appeared last in the questionnaire to avoid biasing the other measures; however, subjects' comments indicated their inability to discriminate their subjective anxiety "in the waiting room" and their subjective anxiety "right now." Since it would be impossible to determine whether the measure represented initial differences or was itself affected by the manipulations and since it therefore would not be a helpful covariate (Sprott, 1970), it was not analyzed and will not be discussed further.

necessary. All manipulations and measures following anesthetic injection took a total of 5-10 minutes, after which the extraction was performed as usual.

After the extraction the operating dental student and assistant were asked for a rating of the subject's anxiety or nervousness based on his observable behavior (except tremor) for the first few minutes after the questionnaire was completed.

(See Appendix A for the consent form and the complete instructions.)

## Procedure

### Independent variables

Three variables were manipulated in a factorial design. The design is illustrated in Figure 1. Within each sex the subjects were assigned sequentially to conditions (except for Actual Arousal to which the experimenter remained blind). This served to keep the number of males and females equal in each cell in case of differences in their responses to the conditions. Sequential assignment assured that the groups were formed in an unbiased manner (Underwood, 1966) and spread the effects of any changes in clinic procedure and personnel (who changed every week) over all conditions. When every cell contained at least four males and four females, extra males and females were chosen at random in the larger cells and excluded from the analyses.

Actual arousal. Actual autonomic arousal was manipulated by the

| Attribution of Arousal | | Extrinsic | | | Intrinsic | | |
|---|---|---|---|---|---|---|---|
| Cognition of Arousal | | No Information | Unaroused | Aroused | No Information | Unaroused | Aroused |
| Actual Arousal | Epinephrine | | | | | | |
| | No Epinephrine | | | | | | |

Figure 1. Schematic Representation of the Design.

use of different drugs for the local anesthetic injection. The anesthetic used most often in the Oral Surgery Clinic is lidocaine hydrochloride[1] in a two-percent solution with epinephrine 1:100,000. Two-percent lidocaine hydrochloride without epinephrine is also used occasionally. The aid of the dental students was enlisted; they were told that it was important that the study have equal numbers of patients anesthetized initially with epinephrine and without epinephrine, and important that the two groups not differ in the severity of the procedures to be performed. For each subject the operating dental student chose one of these two anesthetics without informing the experimenter.

Anesthesia for extraction of mandibular teeth is obtained by blocking the generation and transmission of nerve impulses in the inferior alveolar branch of the mandibular nerve. To accomplish this, the dentist injects the anesthetic near the mandibular foramen on the inside of the mandible at the rear of the mouth. Anesthetic injected at this site on the side of the mouth anesthetizes all the teeth and the jaw on that side. In addition, a much smaller amount is injected into the cheek nearby for infiltration anesthesia of the long buccal branch of the mandibular nerve. The standard dental cartridge contains 1.8 cubic centimeters; in this study one to two cartridges were injected before the questionnaire and

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[1] Also known by trade names, such as xylocaine and octocaine.

other measures.[1]

Epinephrine is included in dental anesthetics because it produces local vasoconstriction which keeps the anesthetic from being carried away from the injection site and thereby prolongs the anesthesia. One cubic centimeter of two-percent lidocaine with epinephrine 1:100,000 contains 0.02 grams of lidocaine and 0.00001 grams or 10 micrograms of epinephrine. The amount injected in this study was between one and two cartridges or 18-36 micrograms. In contrast, Schachter (Schachter & Singer, 1962) used subcutaneous injections of one cubic centimeter of 1:1,000 epinephrine, that is, 0.001 grams or 1000 micrograms (1 milligram). Injections in the present study were not intravenous; the dental students routinely used aspirating syringes and aspirated before injection in order to insure that the tip of the needle was not in a blood vessel. Many dental sources (Cheraskin & Prasertsuntarasai, 1957, 1958, 1959; and Glover, 1968, among others) argue that, unless injection is intravascular, the systemic effects of the amounts of epinephrine injected in dentistry are negligible, even when compared to plain lidocaine which tends to be a cardiac depressant (Goodman & Gilman, 1970). Since the intent was to compare perceptible differences in Actual Arousal in this study, a pilot study was undertaken (Appendix B). Ten subjects were injected with lidocaine and with lidocaine with epinephrine on separate

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[1] Further injections were often necessary because the block was missed or the anesthesia had worn off, but of course these occurred during the actual extraction, too late to affect the dependent measures.

occasions. Data from one subject had to be discarded. Seven of the remaining nine chose epinephrine as the more arousing drug; the difference in reported autonomic arousal approached significance on a one-tailed t test for paired observations ($p < 0.06$). So Actual Arousal was manipulated by administering lidocaine with epinephrine in the Epinephrine condition and lidocaine without epinephrine in the No Epinephrine condition.

Attribution of arousal. Following the post-injection measurement of pulse rate and blood pressure, one-half of the Epinephrine subjects and one-half of the No Epinephrine subjects were given the Extrinsic Attribution manipulation. The other half constituted the Intrinsic Attribution group. The Extrinsic subjects were casually told that any arousal which they felt was probably due to the drug injected. The communication was worded carefully so as not to imply that the subject necessarily was aroused:

> You know, some people notice some side effects from the drug used to numb their mouth. They feel their heart pounding or their hands shaking or other such changes. These changes are harmless but sometimes quite noticeable.

The Intrinsic subjects were told nothing about the source of any arousal. One advantage of using dental anesthetics to manipulate autonomic arousal is that the presence of epinephrine in some anesthetic solutions is not commonly known, or suspected after injection. Subjects who had no alternative explanation for any arousal of which they were aware therefore were expected to make an intrinsic attribution, attributing their

arousal to anticipation of the dental treatment.

Cognition of degree of arousal. Cognition of Arousal was manipulated immediately after Attribution. This factor was divided into three conditions. One-third of the subjects in each condition of the other factors were given the Unaroused manipulation regardless of the pulse rate and blood pressure which had just been measured. They were casually told:

> Your heart rate and blood pressure haven't changed much at all and they weren't very high compared to most people's here anyway.

One-third of the subjects in each condition of the other factors were given No Information about their degree of arousal. This condition was included to have some subjects in which any natural perception of arousal was neither reinforced nor contradicted by Cognition of Arousal.

Regardless of their actual pulse rate and blood pressure, one-third of the subjects in each condition of the other factors were casually told they were Aroused as follows:

> Your heart rate and blood pressure have gone up some. They are higher than lots of people's here but nothing to worry about.

Manipulation checks

Severity of dental procedure. Lidocaine without epinephrine provides operating anesthesia of shorter duration than lidocaine with epinephrine; in addition, extraction produces more bleeding when there is no vasoconstrictor in the solution. The operating dental students were

informed that they could administer a different anesthetic if necessary after the experimenter talked with the patient and were asked not to let the number of teeth to be pulled affect their choice of anesthetic for the initial injection. Nevertheless, it was possible that Epinephrine and No Epinephrine subjects would differ in the number of teeth which they expected to have extracted and that this expectation would affect the dependent variables. So, the number of teeth to be extracted was recorded for each subject.

Amount of anesthetic. As stated in connection with the Actual Arousal factor, subjects were injected with 1-2 cartridges of anesthetic. Amount of anesthetic was recorded for each subject in order to determine whether there were any systematic differences between groups. The amount of anesthetic injected was estimated from the anesthetic solution remaining in the used cartridge(s). This was a rough estimate, first, because the cartridges were not graduated and, second, because a small, but variable, fraction was expelled in preparing the syringe.[1]

Autonomic measures. Pulse rate and blood pressure were measured approximately one minute after injection as manipulation checks on

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[1] Standardizing the amount injected would not have kept the functional amount of lidocaine or epinephrine (within the Epinephrine condition) constant; the effect could still vary according to body weight, and due to the exact site of injection and migration of the solution. Galbreath and Eklund (1970) have discussed migration of lidocaine with epinephrine from particular injection sites in relation to the effects of lidocaine; such anatomical differences are probably relevant to epinephrine effects as well.

the Actual Arousal manipulation. Pulse rate was counted by palpating the radial artery and counting the pulse for 15 seconds; this number was converted to beats per minute for the analysis.[1] Systolic and diastolic blood pressure were measured indirectly at the brachial artery with an aneroid sphygmomanometer and stethoscope; disappearance of the Korotkoff sounds, rather than muffling, was used as diastolic pressure. The American Heart Association's suggestions for technique were followed (Kirkendall, Burton, Epstein, & Freis, 1967). All pulse rate and blood pressure measures were taken by the experimenter with the subject in the same position and on the same arm. Pulse rate was always measured prior to the blood pressure.

Although continuous electrophysiological recording of autonomic measures would have been more informative as far as the autonomic effect of the epinephrine was concerned, the appropriate facilities were not readily available; and such recording would have required substantial disruption of clinic routine. Manual determination of pulse rate and blood pressure had several important advantages. Pulse and blood pressure are normally determined in this manner before oral surgery; so the only

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[1] Although this seems like a rather short time period, the effects of epinephrine were expected to be quite transient. Vernale (1960) found greater change in pulse and blood pressure 30 seconds or 60 seconds after dental injection of lidocaine with epinephrine than was found at 5 minutes post-injection. Such transience was not expected to prevent the cue function of arousal, but it did necessitate measuring pulse rate quickly in order to get to the more time-consuming blood pressure measurement and the subsequent manipulations while arousal was still being experienced.

change in clinic routine was the repetition of these measures after the injection. Moreover, since these measures were routine, they were unobtrusive and would not in themselves predispose subjects to think a physiological change was expected. In addition, the subjects' familiarity with the measures was essential to the Cognition of Arousal manipulation.

The subject rested in the dental chair for about 10 minutes while his medical history was taken, the planned oral surgery was approved, and the room was prepared. Pulse rate and blood pressure were then taken before the anesthetic injection. These were the baseline or Pre-Injection measures. The Post-Injection measures were taken approximately one minute after the injection was completed. Each Pre-Injection measure was used as the covariate for the corresponding Post-Injection score in order to correct the Post-Injection measures for any dependence on their initial levels.[1]

Autonomic report. Subjects were given a questionnaire after the Attribution and Cognition of Arousal manipulations. (For the complete questionnaire, see Appendix C.) Following the dependent measures in the questionnaire were five items on which the subject was asked to rate the degree to which he had felt his heart pounding, tremor, increased

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[1] Autonomic measures are generally correlated with pre-stimulus values due to the individual's tendency to show his characteristic level on that variable and due to the Law of Initial Values (Wilder, 1956, 1965). Like Wilder, Benjamin (1963, 1967) argues that difference scores generally are not independent of their initial levels; she suggests Analysis of Covariance as the best method for a statistical test which is corrected for initial levels.

heart rate, increased depth of respiration, or increased rate of respiration on a 0 to 3 scale. The subject was asked to report his perceptions since the first blood pressure measure rather than since the injection in order to avoid artifactual induction of an extrinsic attribution before the manipulation check for the Attribution factor.

The report of autonomic changes was a manipulation check for the Actual Arousal factor. On the basis of the pilot study, the Epinephrine condition was expected to produce greater report of perceived autonomic changes.

This measure was also included as a manipulation check for the Cognition of Arousal factor. A lack of significant differences between the Aroused and No Information conditions would be inconclusive because one may believe that his heart rate and blood pressure have increased without actually perceiving a change in arousal; however, no significant difference for Unaroused versus No Information would indicate failure of that aspect of the manipulation. In either case significant differences would indicate that the manipulation was effective.

Attribution of reported autonomic changes. One open-ended and four specific items were included in the questionnaire after the Autonomic Report scale as a manipulation check for the Attribution factor. The open-ended item was presented before the specific alternatives were seen in case some plausible source of arousal had been omitted. On the specific items the subject reported how much of his arousal he attributed to

anticipation of the dental work, the drug, answering the questionnaire, and pain from the injection. Obviously, only subjects who had reported some arousal could answer these questions.

Dependent variables

Post-manipulation autonomic measures. After the Attribution and Cognition of Arousal manipulations, pulse rate and blood pressure were measured again, by the methods described above. The purpose of this measure was to ascertain whether the manipulations, especially Cognition of Arousal, had autonomic effects. The actual Post-Injection measures were used as covariates for the Post-Manipulation measures.

Subjective report of anxiety. This was the first scale on the questionnaire and therefore was administered just after the Post-Manipulation autonomic measures. The subject was instructed that the questions referred to his feelings at the present. Three bipolar items were used; these had been constructed from six items on the state section of the State-Trait Anxiety Inventory (Spielberger, Gorsuch, & Lushene, 1968). While the measures were being designed, this 20-item scale was administered to about 20 patients awaiting oral surgery. Many did not understand the word "anxious" or several other items on the test. The six pairs which were judged to be most sensitive were Tense-Relaxed, Nervous-Calm, and Jittery-At Ease. The labels for the points on the scale were also changed to terms which were more familiar to the patients.

In addition, many patients given the whole Spielberger inventory rated themselves as feeling extremely pleasant or joyful. After the surgery was completed, many of these same patients admitted that they were quite nervous but embarrassed to admit it. This study therefore included assurances of confidentiality in the consent form and in the instructions preceding the questionnaire. Subjects were also told that many people were uncomfortable in this situation and that their participation could only help other patients if they answered honestly (Appendix A).

Tendency to avoid. This is what McGrath (1970) calls a self-report measure of behavior. Instead of using deception, the question was worded hypothetically and the subject was asked to predict what other people would do based on his own reaction. The subject was asked whether people would return to be in an experiment which would be very much like his experience today and if so how much they would have to be paid to return. The second question was included in order to obtain a quantity more amenable to statistical analysis. Whether the subject predicted that people would not return or estimated how much they would have to be paid, he was asked to state the reason for his prediction.

Observed behavior. The other behavioral measure was an observational measure. The operating dental student and his assistant were asked to rate how nervous or anxious their patient was when they re-entered the operatory after the patient's interview with the experimenter. They were asked to disregard any tremor since it could be due to the drug and to

disregard his behavior after the extraction began since subsequent responses could be due to pain or their own technique. The raters were allowed to use their own criteria for nervous or anxious behavior. They were not aware of the subject's responses on the questionnaire items. Although the raters did know the drug condition, they were not sympathetic to the hypothesis that the epinephrine in dental anesthetic could affect the patient autonomically or behaviorally.

This measure has limitations and must be interpreted cautiously. The raters were not trained observers, and no reliability check was possible since subjects were rated by different pairs of observers. To some extent, the rating may be a measure of the rater's powers of observation. Nevertheless, it was the only feasible means of assessing the subject's behavior in the dental chair, and a rating by the operating dentist and his assistant was of special interest since they are usually the only people who can assess the patient's behavior and respond to it.

# RESULTS

## Methods of Analysis

The analysis of variance (ANOVA) and analysis of covariance (ANACOVA) were used to analyze most of the dependent measures and manipulation checks. One exception was the Attribution manipulation check which was analyzed by chi-square, as explained below under the Attribution factor. Another exception was one of the main dependent measures, Tendency to Avoid a similar situation, which could not be analyzed using the analysis of variance because many subjects proved to be unable or unwilling to state a fair wage for coming back to be in a dental experiment. The binary choice as to whether people would Return or not (Avoid) was therefore analyzed by chi-square tests.

Pulse rate was analyzed in beats per minute and systolic and diastolic blood pressure as millimeters of mercury. The levels to be analyzed were adjusted by the pre-stimulus level of the same autonomic measure for the ANACOVA. Each subject's total on the Subjective Report scale was used, with scores on the "calm," "relaxed," or "at ease" side of the midpoint given a negative sign. The Autonomic Report score was also the sum of all items on the scale. The ratings of the two judges on

the Observed Behavior item were averaged, and that average was analyzed for each subject. Cell means for variables analyzed by ANOVA, adjusted cell means for variables analyzed by ANACOVA, and frequency per cell for variables analyzed by chi-square are presented in Appendix E.

All ANOVAs and ANACOVAs were performed with the Multivariate Analysis of Variance (MANOVA) computer program from the L. L. Thurstone Psychometric Laboratory at the University of North Carolina at Chapel Hill. The MANOVA itself analyzes an optimally weighted linear combination of the variates. The multivariate test is Wilk's lambda criterion (likelihood ratio test) using Rao's approximate F test. A significant or high multivariate F indicates that a significant univariate F (using one of the same variates) for the same effect is not a spurious result.

Unfortunately, only two dependent variables could be analyzed by MANOVA in this study. As stated above, Tendency to Avoid required analysis by a nonparametric test; in addition, the use of covariates in the analyses of Post-Manipulation Pulse Rate, Systolic Blood Pressure, and Diastolic Blood Pressure precluded their inclusion with other measures for which the covariates were not appropriate. Subjective Report and Observed Behavior were analyzed by MANOVA, and the results are presented below with the appropriate factors. Only two multivariate Fs were significant, but three others were high enough to lend credence to

significant univariate tests. Since all of the relevant variables could not be included in the MANOVA tests, and since there are replications in the literature for many of the effects tested, univariate tests (ANOVA) are reported even where the multivariate test was not significant.

Tests of simple effects within one level of a factor, simple interactions, etc., were also analyzed by ANOVA using the univariate tests in the MANOVA program; all tests used the within-cells variation from all the cells in the design to estimate the error term.[1]

The MANOVA, ANOVAs, and ANACOVAs were actually based on a four-factor design. Since it was necessary to use both male and female subjects, the analyses were performed with Sex included as a fourth factor to determine whether a significant amount of variance was due to Sex. It was found that the multivariate $\underline{F}$ was highly significant for Sex ($p < .001$). In addition, two univariate main effects were significant. Women scored higher on Subjective Report of anxiety ($p < .048$) and also on Observed Behavior ($p < .001$). Moreover, there were significant interactions involving Sex on Subjective Report, Autonomic Report, adjusted Post-Manipulation Pulse Rate, and adjusted Post-Manipulation Diastolic Blood Pressure. An interaction indicates that one sex is not simply

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[1] The analyses discussed below which used Amount of Anesthetic as a covariate were exceptions. These tests used the within-cell error from only the Epinephrine cells for comparisons within that condition and the within-cells error from the No Epinephrine cells for comparisons within that condition because the covariates were different anesthetics in the two conditions.

higher than the other sex on a certain variable; rather, the effects of other independent variables are different in degree and/or in direction for Female subjects than for Male subjects. Because of the interactions with Sex, and in order to reduce the within-cells variance, the four-factor analysis was retained.

## Preliminary Analyses

In using a real-life stress, there were several potential sources of error which could not be tightly controlled. Ideally, unbiased assignment of subjects to cells would have prevented any significant differences on Severity of oral surgery and differences within the Epinephrine or No Epinephrine conditions on Amount of Anesthetic (especially important in the Epinephrine cells), and Post-Injection (pre-manipulation) Pulse Rate and Blood Pressure.

### Severity

An ANOVA on Severity (number of teeth to be extracted) revealed no significant main effects or interactions (Table 21, Appendix D). .

### Amount of anesthetic

Since two different anesthetics were used, Amount of Anesthetic (represented as 1.0 to 2.0 cartridges) was analyzed in one 12-cell ANOVA for the No Epinephrine cells. There were significant differences within these analyses (Tables 22a and 22b, Appendix D). This might suggest

the use of Amount of Anesthetic as a covariate for the dependent measures and manipulation checks to reduce the error term for these measures by removing any variation linearly associated with differences in Amount of Anesthetic. However, since the covariate would be the amount of a different anesthetic in each condition of Actual Arousal, this solution would preclude analyzing all 24 cells together and testing the Actual Arousal main effect and interactions.

In addition, there is evidence that such an adjustment is not necessary. First, the significant Amount of Anesthetic effects were not generally in the direction expected for the manipulation checks or dependent measures (Tables 23a, 23b, and 23c, Appendix D). Second, of the twelve tests for the regression of Post-Injection autonomic measures, Subjective Report, Observed Behavior, or Autonomic Report on Amount of Anesthetic, the pooled within-cells regression was significantly different from zero only for Post-Injection Pulse Rate ($p < .024$) and Subjective Report ($p < .039$) within the Epinephrine condition. Surprisingly, both of these regressions were negative, indicating that greater Amount of Anesthetic was associated with lower Post-Injection Pulse Rate and Subjective Report.[1] For each of these measures, analyses with and without Amount

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[1] The within-cells raw score regression coefficient for Post-Injection Pulse Rate on Amount of Anesthetic was -12.174 and for Subjective Report on Amount of Anesthetic was -3.896. Again, note that the raw scores for Amount of Anesthetic are in number of cartridges rather than in cubic centimeters.

of Anesthetic as a covariate were performed for purposes of comparison (Tables 24a, 24b, 25a, 25b, Appendix D).[1] The same tests approached significance in the case of Post-Injection Pulse Rate and were significant in the case of Subjective Report. The p values of these effects were similar whether the covariate was used or not, and the direction or form of the effects was the same. Furthermore, the error term for these analyses was not substantially changed by the addition of Amount of Anesthetic as a covariate. Because of the inconsequential differences between the analyses with Amount of Anesthetic as a covariate and those without it, the vagaries of administration of the anesthetic, and the inaccuracies in measurement of Amount of Anesthetic, Amount of Anesthetic was not used as a covariate in the main analyses below and the 24-cell, four-factor analyses were possible.

## Post-injection autonomic measures

A similar problem arose because there were significant Post-Injection differences on the autonomic measures within each Actual Arousal condition even after they were adjusted for Pre-Injection levels by ANACOVA.[2] There were differences on adjusted Post-Injection Pulse

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[1] The equality of the within-cells regressions for all cells of the analysis was tested for every ANACOVA reported in this paper with the exception of those using three covariates (too few degrees of freedom in the error term for the test). No violations of the assumption of homogeneity were found.

[2] There were also significant differences on Pre-Injection measures (Tables 26a, 26b, 26c, Appendix D). Some caution is necessary in using

Rate within the Epinephrine and No Epinephrine conditions and on adjusted Post-Injection Systolic Blood Pressure within the Epinephrine condition, but none on adjusted Post-Injection Diastolic Blood Pressure (Tables 27a, 27b, 27c, 28a, 28b, and 28c, Appendix D).

However, adjusting Subjective Report, Observed Behavior, and Autonomic Report for these differences in Post-Injection autonomic measures would also correct for the difference between the Actual Arousal conditions, not just the differences within the Actual Arousal conditions. To determine whether this "solution" was necessary, analyses with and without the Post-Injection autonomic measures were compared, within the Epinephrine and No Epinephrine conditions, for the two variables showing a significant within-cells regression on the three Post-Injection autonomic measures: Subjective Report ($p < .031$) and Observed Behavior ($p < .008$).[1], [2] The same effects were significant at the .050 level or

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ANACOVA when there are significant differences on the covariate, although this is when ANACOVA is needed most (Cochran, 1957). The primary danger is that the regression equation may not hold in areas of extrapolation; this was not considered to be a problem with the present data because the scores in different cells overlapped.

[1] The unadjusted Post-Injection autonomic measures were used as the covariates because these measures included the effect of the initial (Pre-Injection) levels, as well as the differences on Post-Injection levels.

[2] When all three covariates were used together, the within-cells raw regression coefficients for Subjective Report were .078 for the regression on Post-Injection Pulse Rate, .026 for the regression on Post-Injection Systolic Blood Pressure, and -.093 for the regression on Post-Injection Diastolic Blood Pressure. The comparable values for Observed Behavior were .016 for Pulse Rate, .011 for Systolic Blood Pressure, and -.022 for Diastolic Blood Pressure.

better in both cases, with the exceptions of two interactions which were significant without the covariates but not with them (Tables 29a, 29b, 30a, 30b, 31a, 31b, 32a, 32b, Appendix D). However, both of these interactions had a p value of less than .109 and the forms of the interactions were similar with and without the covariates. The other significant effects were in the same direction or form in every case. The error mean squares were also very close whether the covariates were used or not.

Because of the importance of testing the Actual Arousal effects in this experiment and because there was no evidence of substantial changes when the same effects were analyzed using Amount of Anesthetic or Post-Injection autonomic measures as covariates, ANACOVA was used only for the analyses of Post-Injection and Post-Manipulation autonomic measures. Relative confidence can be placed in the direction and significance (although not the exact level) of the effects reported below.[1]

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[1] The significant effects discussed above have been presented very briefly because the concern was with finding valid and useful statistical procedures rather than with interpreting the data. Some of the effects tested within the Epinephrine or No Epinephrine cells above are not reported in the results below because it would not have been statistically valid to do so. When the whole four-factor, 24-cell design is considered together, these are simple effects (tests within one condition of a factor). Simple effects are generally tested only if they are relevant to a specific hypothesis or if there are significant overall interactions, indicating that the effect of one factor differs within the levels of another factor. In addition, the simple effects tested below were all compared to the mean square error term from the whole design; the effects reported above in the discussion of Amount of Anesthetic as a possible covariate were tested against the mean square error appropriate to a smaller design of only the Epinephrine cells or No Epinephrine cells and therefore may vary from the corresponding tests below.

## Effectiveness of the Manipulations

### Actual arousal manipulation checks

An ANACOVA with Post-Injection autonomic measures adjusted for Pre-Injection level on the same autonomic measure demonstrated that the Epinephrine and No Epinephrine groups did differ in actual autonomic arousal (Tables 1, 2, and 3). There was a significant main effect for the Actual Arousal factor on adjusted Post-Injection Pulse Rate ($p < .001$) and Diastolic Blood Pressure ($p < .003$), but not on Systolic Blood Pressure. Pulse Rate averaged 6.263 beats per minute higher and Diastolic Blood Pressure averaged 5.155 millimeters of mercury lower in the Epinephrine group. None of the tests for other main effects or interactions were significant for the measures of autonomic arousal when the whole 24-cell design was considered together.

The Autonomic Report measure indicated that the manipulation was also effective in producing perceptible differences in arousal. In fact, 40 Epinephrine subjects (out of 48) as compared to 26 No Epinephrine subjects reported some arousal on the Autonomic Report scale. However, when the actual scores were analyzed by ANOVA the differences held up only for Females. Although there was a significant main effect for Actual Arousal with Epinephrine subjects reporting more autonomic arousal than No Epinephrine subjects ($p < .009$), there was also a significant Actual Arousal X Sex interaction ($p < .007$; Tables 4 and 5). Only Females reported more autonomic arousal in the Epinephrine condition

## Table 1

Summary of Analysis of Covariance: Post-Injection Pulse Rate with Pre-Injection Pulse Rate as a Covariate as a Function of All Independent Variables

| Source | df | MS | F | p |
|---|---|---|---|---|
| Actual Arousal (A) | 1 | 940.000 | 21.429 | .001 |
| Attribution (B) | 1 | 18.539 | < 1 | |
| Cognition of Arousal (C) | 2 | 26.404 | < 1 | |
| Sex (D) | 1 | 8.449 | < 1 | |
| A X B | 1 | 60.680 | 1.383 | n.s. |
| A X C | 2 | 13.240 | < 1 | |
| A X D | 1 | 13.918 | < 1 | |
| B X C | 2 | 16.820 | < 1 | |
| B X D | 1 | 1.578 | < 1 | |
| C X D | 2 | 92.439 | 2.107 | n.s. |
| A X B X C | 2 | 2.605 | < 1 | |
| A X B X D | 1 | 1.352 | < 1 | |
| A X C X D | 2 | 66.426 | 1.514 | n.s. |
| B X C X D | 2 | 72.063 | 1.643 | n.s. |
| A X B X C X D | 2 | 127.062 | 2.897 | .062 |
| Within-cells regression | 1 | 8289.574 | 188.978 | .001 |
| Error | 71 | 43.865 | | |

Table 2

Summary of Analysis of Covariance: Post-Injection Systolic Blood Pressure with Pre-Injection Systolic Blood Pressure as a Covariate as a Function of All Independent Variables

| Source | df | MS | F | p |
|---|---|---|---|---|
| Actual Arousal (A) | 1 | 4.016 | < 1 | |
| Attribution (B) | 1 | 8.199 | < 1 | |
| Cognition of Arousal (C) | 2 | 83.535 | 2.436 | .095 |
| Sex (D) | 1 | 28.758 | < 1 | |
| A X B | 1 | 3.168 | < 1 | |
| A X C | 2 | 57.139 | 1.666 | n. s. |
| A X D | 1 | .309 | < 1 | |
| B X C | 2 | 14.002 | < 1 | |
| B X D | 1 | 1.539 | < 1 | |
| C X D | 2 | 28.756 | < 1 | |
| A X B X C | 2 | 9.041 | < 1 | |
| A X B X D | 1 | .359 | < 1 | |
| A X C X D | 2 | 45.684 | 1.332 | n. s. |
| B X C X D | 2 | 7.580 | < 1 | |
| A X B X C X D | 2 | 41.527 | 1.211 | n. s. |
| Within-cells regression | 1 | 9259.859 | 269.982 | .001 |
| Error | 71 | 34.298 | | |

### Table 3

Summary of Analysis of Covariance: Post-Injection Diastolic Blood Pressure with Pre-Injection Diastolic Blood Pressure as a Covariate as a Function of All Independent Variables

| Source | df | MS | F | p |
|---|---|---|---|---|
| Actual Arousal (A) | 1 | 629.203 | 9.358 | .003 |
| Attribution (B) | 1 | 9.988 | < 1 | |
| Cognition of Arousal (C) | 2 | 80.555 | 1.198 | n. s. |
| Sex (D) | 1 | .660 | < 1 | |
| A X B | 1 | 39.250 | < 1 | |
| A X C | 2 | 17.682 | < 1 | |
| A X D | 1 | 88.828 | 1.321 | n. s. |
| B X C | 2 | 145.244 | 2.160 | n. s. |
| B X D | 1 | 115.555 | 1.719 | n. s. |
| C X D | 2 | 10.014 | < 1 | |
| A X B X C | 2 | 75.619 | 1.125 | n. s. |
| A X B X D | 1 | 57.535 | < 1 | |
| A X C X D | 2 | 100.004 | 1.487 | n. s. |
| B X C X D | 2 | 10.146 | < 1 | |
| A X B X C X D | 2 | 4.633 | < 1 | |
| Within-cells regression | 1 | 5436.191 | 80.852 | .001 |
| Error | 71 | 67.236 | | |

### Table 4

Summary of Analysis of Variance: Autonomic Report as a Function of Actual Arousal and Cognition of Arousal and Their Interactions

| Source | df | MS | F | p |
|---|---|---|---|---|
| Actual Arousal (A) | 1 | 32.667 | 7.204 | .009 |
| Cognition of Arousal (C) | 2 | 32.156 | 7.091 | .002 |
| A X Attribution (B) | 1 | 12.042 | 2.655 | n.s. |
| A X C | 2 | 4.198 | < 1 | |
| A X Sex (D) | 1 | 35.042 | 7.727 | .007 |
| B X C | 2 | .323 | < 1 | |
| C X D | 2 | .698 | < 1 | |
| A X B X C | 2 | 9.698 | 2.139 | n.s. |
| A X C X D | 2 | 6.698 | 1.477 | n.s. |
| B X C X D | 2 | 7.406 | 1.633 | n.s. |
| A X B X C X D | 2 | .406 | < 1 | |
| Error | 72 | 4.535 | | |

than in the No Epinephrine condition ($p < .001$). There were no other interactions involving Actual Arousal on this measure.

Table 5

Autonomic Report by Actual Arousal and Sex

| | Females | Males |
|---|---|---|
| Epinephrine | 3.542 | 1.875 |
| No Epinephrine | 1.167 | 1.917 |

### Cognition of arousal manipulation check

There was a significant main effect for Cognition of Arousal on Autonomic Report ($p < .002$; Table 4). The subjects who were told they were Aroused reported significantly more autonomic arousal than the subjects who were given No Information ($p < .002$). However, subjects who were told that they were Unaroused did not report significantly less autonomic arousal than No Information subjects. This manipulation was clearly more effective for the Aroused condition. There were no interactions involving Cognition of Arousal on the Autonomic Report measure.

### Attribution manipulation checks

The manipulation check for Attribution consisted of four questions,

given only to subjects who scored higher than zero on Autonomic Report. Only two subjects attributed any of their arousal to answering the questions, an item which was included solely to provide an option unrelated to the hypotheses. Arousal was attributed to pain from the shot by 29 subjects, but a chi-square test did not reveal any significant association between the Attribution conditions and whether any arousal was attributed to pain or not.[1]

Contrary to expectations, the proportion of Intrinsic Attribution subjects who actually expressed an intrinsic attribution (anticipation of dental work) was no greater than the proportion of Extrinsic subjects expressing the intrinsic attribution. The chi-square for Attribution conditions and some dental attribution versus no dental attribution was not significant ($X^2 = .0485$). To a limited extent, the Extrinsic Attribution manipulation was effective in inducing the extrinsic (drug) attribution

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[1] These data were analyzed by chi-square tests because they obviously violated the assumptions of normal distributions of scores and homogeneity of variance which underlie $\underline{F}$ tests, and $\underline{t}$ tests on two or more samples. In actual practice the assumption of normality can be violated with relative impunity with samples of moderate size, and routine tests for homogeneity of variance are not advisable because they often give evidence for unequal variances when inferences about means would be quite valid (Hays, 1963). In general, this assumption can also be violated without serious risk when sample sizes are equal, as in the ANOVAs and ANACOVAs reported in this paper, but not when sample sizes are unequal as on these Attribution manipulation checks. Chi-square tests are more appropriate on these measures for a second reason; a large proportion of subjects gave ratings of either 0 or 1, especially on the questions dealing with attribution to pain or to the drug. The chi-square tests compared the Intrinsic Attribution and Extrinsic Attribution conditions on whether any attribution or no attribution was made to a particular source (pain, dental work, or drug).

intended. The chi-square for Attribution conditions and some drug attribution versus no drug attribution was significant ($p < .005$; Table 6). However, although approximately 80% of the drug attributions were made by Extrinsic subjects, over 50% of the Extrinsic subjects attributed none of their autonomic arousal to the drug injected.

Table 6

Two X Two Contingency Table and Chi-Square for Attribution Factor and Drug Attribution

| | Attribution of Some Arousal to Drug | Attribution of No Arousal to Drug |
|---|---|---|
| Extrinsic | 15 | 19 |
| Intrinsic | 4 | 28 |

$X^2 = 8.039$ $p < .005$

Thus, Extrinsic subjects made more extrinsic (drug) attributions but no fewer intrinsic attributions than Intrinsic subjects. In fact, 10 of the 15 Extrinsic subjects who attributed some of their arousal to the drug also attributed some of their arousal to anticipation of dental work. The Attribution manipulation was effective in inducing some attributional differences, but the differences were not as great as expected.

## Major Findings: Effects of Independent Variables on Dependent Measures

### Actual arousal

Multivariate tests. The main effect for Actual Arousal was not significant when tested by MANOVA with Subjective Report and Observed Behavior as the two variates (see Table 7). Although no MANOVA interactions involving Actual Arousal were significant at the .05 level, the multivariate Fs were high for Actual Arousal X Attribution ($p < .114$) and Actual Arousal X Attribution X Sex ($p < .081$).[1] These two interactions are important in light of the univariate analysis of Subjective Report.

Subjective report. The main effect for Actual Arousal was not significant for the Subjective Report measure. There was a significant Actual Arousal X Attribution X Sex effect ($p < .038$; Tables 8 and 9). This interaction indicates that the effect of Actual Arousal was different depending on the Attribution condition and on Sex. Analysis of the simple effects showed that Subjective Report of anxiety was significantly higher

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[1] A large number of statistical tests were performed to analyze the data from this experiment, increasing the probability that one or more effects might be significant due to chance alone. To avoid further inflation of the probability of a Type I error, marginal effects on the univariate tests are not reported. Only effects of $p < .05$ are reported for the univariate tests. All ps < .10 are reported in the tables for readers who desire this information. The multivariate tests are another matter. Since a high multivariate F is helpful in determining that the corresponding significant univariate effect is not due to chance alone, the five most reliable multivariate effects in Table 7, including three with $p > .05$, are reported.

Table 7

Summary of Multivariate Analysis of Variance[a]: Effects of All Independent Variables with Subjective Report and Observed Behavior as Variates

| Source | df Hypothesis | df Error | F | p |
|---|---|---|---|---|
| Actual Arousal (A) | 2 | 71 | < 1 | |
| Attribution (B) | 2 | 71 | 1.032 | n.s. |
| Cognition of Arousal (C) | 4 | 142 | 4.340 | .002 |
| Sex (D) | 2 | 71 | 5.968 | .004 |
| A X B | 2 | 71 | 2.238 | .114 |
| A X C | 4 | 142 | < 1 | |
| A X D | 2 | 71 | < 1 | |
| B X C | 4 | 142 | 2.208 | .071 |
| B X D | 2 | 71 | < 1 | |
| C X D | 4 | 142 | 1.472 | n.s. |
| A X B X C | 4 | 142 | < 1 | |
| A X B X D | 2 | 71 | 2.608 | .081 |
| A X C X D | 4 | 142 | 1.239 | n.s. |
| B X C X D | 4 | 142 | 1.455 | n.s. |
| A X B X C X D | 4 | 142 | < 1 | |

[a] Using Wilks lambda criteria with Rao's approximate F test.

Table 8

Summary of Analysis of Variance:Effects of All Independent Variables on Subjective Report

| Source | df | MS | F | p |
|---|---|---|---|---|
| Actual Arousal (A) | 1 | 15.042 | < 1 | |
| Attribution (B) | 1 | .042 | < 1 | |
| Cognition of Arousal (C) | 2 | 120.875 | 7.750 | .001 |
| Sex (D) | 1 | 63.375 | 4.063 | .048 |
| A X B | 1 | 57.042 | 3.657 | .060 |
| A X C | 2 | 2.042 | < 1 | |
| A X D | 1 | 1.042 | < 1 | |
| B X C | 2 | 67.166 | 4.306 | .017 |
| B X D | 1 | 22.041 | 1.413 | n.s. |
| C X D | 2 | 38.375 | 2.460 | .093 |
| A X B X C | 2 | 6.542 | < 1 | |
| A X B X D | 1 | 70.042 | 4.491 | .038 |
| A X C X D | 2 | 39.542 | 2.535 | .086 |
| B X C X D | 2 | 31.291 | 2.006 | n.s. |
| A X B X C X D | 2 | 1.167 | < 1 | |
| Error | 72 | 15.599 | | |

for Epinephrine subjects than for No Epinephrine subjects within the Intrinsic Attribution condition (p < .044) but not within the Extrinsic Attribution condition; this was as predicted in Hypothesis 1. There were no significant simple interactions with Actual Arousal within either the Intrinsic or Extrinsic conditions.

Table 9

Subjective Report by Actual Arousal,
Attribution, and Sex

| | Females | | Males | |
|---|---|---|---|---|
| | Extrinsic | Intrinsic | Extrinsic | Intrinsic |
| Epinephrine | 1.917 | .833 | -2.583 | 1.667 |
| No Epinephrine | .750 | 0.000 | .083 | -2.167 |

Further analyses revealed that Hypothesis 1 was supported only for Males. The Actual Arousal X Attribution interaction was significant for Males (p < .006) but not for Females. In this significant simple interaction for Males, Subjective Report was greater in the Epinephrine than in the No Epinephrine condition only within the Intrinsic Attribution condition. For Male Intrinsic Attribution subjects, who were not led to believe that their arousal was caused by the drug, an Epinephrine injection was followed by greater Subjective Report of anxiety than was an

injection with No Epinephrine ($p < .020$). There were no simple main effects for Actual Arousal nor additional simple interactions with Actual Arousal for either Males or Females.

Observed behavior. Neither the overall main effect for Actual Arousal nor any overall interactions involving Actual Arousal were significant for the Observed Behavior measure (Table 10).

Tendency to avoid. As discussed previously, this measure was analyzed by chi-square tests. For the two X two contingency table for Epinephrine versus No Epinephrine subjects and those predicting Return versus those predicting Avoid, the chi-square was not significant (Table 11a). Since this test did not provide a test of interactions, chi-square tests for Epinephrine versus No Epinephrine were run within each condition of the other three factors to see whether Tendency to Avoid was affected by Actual Arousal under any of the circumstances represented. In other words, the same categories were tested within Intrinsic and Extrinsic Attribution conditions, within Aroused, No Information, and Unaroused conditions of Cognition of Arousal and within Females and Males.[1] Although there were more Epinephrine than No Epinephrine subjects in the Avoid category for the test within the Intrinsic condition (Table 11b), none of the chi-square tests for Actual Arousal were significant.

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[1] This procedure was followed for the analyses of Tendency to Avoid for each independent variable.

Table 10

Summary of Analysis of Variance: Effects of All Independent Variables on Observed Behavior

| Source | df | MS | F | p |
|---|---|---|---|---|
| Actual Arousal (A) | 1 | .094 | < 1 | |
| Attribution (B) | 1 | 1.042 | 1.744 | n.s. |
| Cognition of Arousal (C) | 2 | .010 | < 1 | |
| Sex (D) | 1 | 7.042 | 11.791 | .001 |
| A X B | 1 | 0.000 | < 1 | |
| A X C | 2 | .219 | < 1 | |
| A X D | 1 | .375 | < 1 | |
| B X C | 2 | .198 | < 1 | |
| B X D | 1 | .010 | < 1 | |
| C X D | 2 | .073 | < 1 | |
| A X B X C | 2 | .406 | < 1 | |
| A X B X D | 1 | .010 | < 1 | |
| A X C X D | 2 | .406 | < 1 | |
| B X C X D | 2 | 1.323 | 2.215 | n.s. |
| A X B X C X D | 2 | 1.073 | 1.796 | n.s. |
| Error | 72 | .597 | | |

Table 11a

Two X Two Contingency Table and Chi-Square for Actual Arousal and Tendency to Avoid

| | Avoid | Return |
|---|---|---|
| Epinephrine | 19 | 29 |
| No Epinephrine | 17 | 31 |
| $X^2 = .1777$ | | |

Table 11b

Two X Two Contingency Table and Chi-Square for Actual Arousal and Tendency to Avoid Within Intrinsic Attribution Condition

| | Avoid | Return |
|---|---|---|
| Epinephrine | 11 | 13 |
| No Epinephrine | 5 | 19 |
| $X^2 = 3.3750$ $.05 < p < .10$ | | |

Post-manipulation autonomic measures. Each of these measures was analyzed by ANACOVA with the Post-Injection level of the same measure used as the covariate. The Epinephrine and No Epinephrine cells differed on autonomic arousal after the injection; when the Post-Manipulation autonomic measures were adjusted for their differences in initial level by using the Post-Injection measures as covariates, the Epinephrine-No Epinephrine arousal difference due to the injection was also removed.[1] This, of course, was not desirable for tests of Subjective Report, Observed Behavior, and Autonomic Report because it would have eliminated the direct effects of the injections. In the present case, the ANACOVA answers the question "Were there differences between the Epinephrine and No Epinephrine conditions in autonomic arousal after the other manipulations, but differences which were not due to the direct physiological effects of the drugs?"[2]

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[1] Although this is generally considered to be a valid use of ANACOVA and valid interpretation of its effect in this situation (Cochran, 1957; Snedecor & Cochran, 1967; Sprott, 1970), Evans and Anastasio (1968; Harris, Bisbee, & Evans, 1971) argue against the use of ANACOVA when the factor to be tested also affects the covariate and further argue that significant treatment effects may remain after adjustment even when the effects are due to the covariate. The tests on Post-Manipulation autonomic measures involving the Actual Arousal factor are therefore presented with less confidence than the tests on Post-Manipulation arousal for the other independent variables, which occurred after the Post-Injection means and therefore could not have affected them.

[2] As is the nature of ANACOVA, this analysis tested differences for groups which had been statistically matched on the autonomic effects of the injections but not on any effects of the injection which were non-linearly related to the Post-Injection measures (such as gross differences

There were no significant effects on adjusted Post-Manipulation Systolic Blood Pressure (Table 12). The Actual Arousal main effect was significant for adjusted Post-Manipulation Diastolic Blood Pressure ($p < .006$); however, there was also a significant Actual Arousal X Sex interaction for the same effect ($p < .014$), such that adjusted Post-Manipulation Diastolic Blood Pressure was actually lower in the Epinephrine condition than in the No Epinephrine condition only for Males ($p < .001$; Tables 13 and 14). In addition, there was a significant Actual Arousal X Attribution X Sex interaction for adjusted Post-Manipulation Pulse Rate ($p < .034$; Tables 15 and 16). There was no significant difference between Epinephrine and No Epinephrine conditions on adjusted Post-Manipulation Pulse Rate within the Intrinsic or Extrinsic Attribution conditions or for Females or Males, but there was a significant Actual Arousal X Attribution interaction for Males ($p < .023$). For Males in the Intrinsic Attribution condition, Post-Manipulation Pulse Rate was higher in the Epinephrine than in the No Epinephrine condition; for Males in the Extrinsic Attribution condition, Post-Manipulation Pulse Rate was actually lower in the Epinephrine condition than in the No Epinephrine condition.

### Cognition of degree of arousal

Multivariate tests. The main effect for Cognition of Arousal was

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between Epinephrine and No Epinephrine subjects in perception or set as one possibility).

Table 12

Summary of Analysis of Covariance: Effects of All Independent Variables on Post-Manipulation Systolic Blood Pressure with Post-Injection Systolic Blood Pressure as a Covariate

| Source | df | MS | F | p |
|---|---|---|---|---|
| Actual Arousal (A) | 1 | 16.871 | < 1 | |
| Attribution (B) | 1 | 2.465 | < 1 | |
| Cognition of Arousal (C) | 2 | 18.471 | < 1 | |
| Sex (D) | 1 | 22.270 | 1.095 | n. s. |
| A X B | 1 | 18.449 | < 1 | |
| A X C | 2 | 50.928 | 2.504 | .089 |
| A X D | 1 | 23.949 | 1.178 | n. s. |
| B X C | 2 | 8.654 | < 1 | |
| B X D | 1 | 30.234 | 1.487 | n. s. |
| C X D | 2 | 19.863 | < 1 | |
| A X B X C | 2 | 5.250 | < 1 | |
| A X B X D | 1 | .207 | < 1 | |
| A X C X D | 2 | 6.697 | < 1 | |
| B X C X D | 2 | 12.723 | < 1 | |
| A X B X C X D | 2 | 4.092 | < 1 | |
| Within-cells regression | 1 | 9774.992 | 480.703 | .001 |
| Error | 71 | 20.335 | | |

## Table 13

## Summary of Analysis of Covariance: Effects of All Independent Variables on Post-Manipulation Diastolic Blood Pressure with Post-Injection Diastolic Blood Pressure as a Covariate

| Source | df | MS | F | p |
|---|---|---|---|---|
| Actual Arousal (A) | 1 | 232.042 | 7.967 | .006 |
| Attribution (B) | 1 | 3.445 | < 1 | |
| Cognition of Arousal (C) | 2 | 20.357 | < 1 | |
| Sex (D) | 1 | 31.992 | 1.098 | n. s. |
| A X B | 1 | 18.871 | < 1 | |
| A X C | 2 | 78.719 | 2.703 | .074 |
| A X D | 1 | 185.141 | 6.356 | .014 |
| B X C | 2 | 76.467 | 2.625 | .079 |
| B X D | 1 | 9.184 | <1 | |
| C X D | 2 | 1.537 | <1 | |
| A X B X C | 2 | 6.889 | <1 | |
| A X B X D | 1 | 10.004 | <1 | |
| A X C X D | 2 | 38.342 | 1.316 | n. s. |
| B X C X D | 2 | 48.885 | 1.678 | n. s. |
| A X B X C X D | 2 | 32.469 | 1.115 | n. s. |
| Within-cells regression | 1 | 5631.934 | 193.356 | .001 |
| Error | 71 | 29.127 | | |

Table 14

Post-Manipulation Diastolic Blood Pressure with Post-Injection Diastolic Blood Pressure as a Covariate by Actual Arousal and Sex

| | Females | Males |
|---|---|---|
| Epinephrine | 72.431 | 68.456 |
| No Epinephrine | 72.896 | 74.553 |

Table 15

Post-Manipulation Pulse Rate with Post-Injection Pulse Rate as a Covariate by Actual Arousal, Attribution, and Sex

| | Females | | Males | |
|---|---|---|---|---|
| | Extrinsic | Intrinsic | Extrinsic | Intrinsic |
| Epinephrine | 83.238 | 81.913 | 78.885 | 84.690 |
| No Epinephrine | 82.820 | 83.893 | 82.478 | 80.747 |

Table 16

Summary of Analysis of Covariance: Effects of All Independent Variables on Post-Manipulation Pulse Rate with Post-Injection Pulse Rate as a Covariate

| Source | df | MS | F | p |
|---|---|---|---|---|
| Actual Arousal (A) | 1 | 2.004 | < 1 | |
| Attribution (B) | 1 | 20.629 | < 1 | |
| Cognition of Arousal (C) | 2 | 1.764 | < 1 | |
| Sex (D) | 1 | 32.418 | 1.029 | n.s. |
| A X B | 1 | 39.473 | 1.253 | n.s. |
| A X C | 2 | 18.588 | < 1 | |
| A X D | 1 | 5.461 | < 1 | |
| B X C | 2 | 10.652 | < 1 | |
| B X D | 1 | 27.801 | < 1 | |
| C X D | 2 | 5.189 | < 1 | |
| A X B X C | 2 | 28.244 | < 1 | |
| A X B X D | 1 | 148.000 | 4.699 | .034 |
| A X C X D | 2 | 8.691 | < 1 | |
| B X C X D | 2 | 8.785 | < 1 | |
| A X B X C X D | 2 | 6.434 | < 1 | |
| Within-cells regression | 1 | 8027.609 | 254.858 | .001 |
| Error | 71 | 31.498 | | |

highly significant for the MANOVA test using Subjective Report and Observed Behavior as the variates ($p < .002$). The Cognition of Arousal X Attribution interaction approached significance ($p < .071$); no other interactions with Cognition of Arousal yielded high multivariate Fs (Table 7, p. 71).

Subjective report. The main effect due to Cognition of Arousal was highly significant ($p < .001$) with the Aroused group highest on Subjective Report of anxiety followed by the No Information group and then the Unaroused group. The Aroused subjects' Subjective Report was significantly higher than the No Information subjects' ($p < .011$). The No Information and Unaroused conditions did not differ significantly, which was not surprising since they did not differ significantly on Autonomic Report. As implied by Hypothesis 2, there was a significant Cognition of Arousal X Attribution interaction ($p < .017$; Table 8, p. 72, and Table 17). In both Attribution conditions the means fell in the order given above, but the

Table 17

Subjective Report by Cognition of Arousal and Attribution

| | Extrinsic | Intrinsic |
|---|---|---|
| Unaroused | -.688 | -2.563 |
| No Information | .313 | -1.063 |
| Aroused | .500 | 3.875 |

simple main effect for Cognition of Arousal was significant only within the Intrinsic Attribution condition. Thus, as predicted by Hypothesis 2, subjects who were induced to believe that they were Aroused exhibited greater Subjective Report of anxiety than subjects given No Information, but only when they were not told that their arousal was due to a drug ($p < .001$). The prediction that telling subjects in the Intrinsic condition that they were relatively Unaroused would result in significantly less Subjective Report than No Information was not borne out by these data. There were no interactions involving Cognition of Arousal within the Intrinsic Attribution condition.

Observed behavior. There was no significant overall main effect for Cognition of Arousal nor were there any significant interactions involving Cognition of Arousal on this dependent variable (Table 10, p. 75).

Tendency to avoid. There were no significant associations between Cognition of Arousal condition and Tendency to Avoid, as tested by chi-square. Table 18 illustrates that a slightly higher proportion of subjects predict Return in the Unaroused condition than in the No Information or Aroused conditions, but the chi-square does not approach significance for this overall test or for the tests within the Epinephrine or No Epinephrine conditions, the Intrinsic or Extrinsic Attribution conditions, or for Females or Males.

Post-manipulation autonomic measures. The significant effects of Cognition of Arousal on Subjective Report did not seem to depend on actual

changes in pulse rate or blood pressure. As predicted in Hypothesis 4, neither the main effect for Cognition of Arousal nor any interactions with Cognition of Arousal were significant for any adjusted Post-Manipulation measure (Table 11, p. 76; Table 12, p. 79; Table 14, p. 81).

Table 18

Three X Two Contingency Table and Chi-Square for
Cognition of Arousal and Tendency to Avoid

| | Avoid | Return |
|---|---|---|
| Unaroused | 9 | 23 |
| No Information | 13 | 19 |
| Aroused | 14 | 18 |
| $X^2 = 1.8666$ | | |

Attribution of arousal

Multivariate tests. The main effect for Attribution was not significant on the MANOVA with Subjective Report and Observed Behavior as the variates. As reported in connection with Actual Arousal and Cognition of Arousal, there were relatively high multivariate F values for Attribution X Actual Arousal ($p < .114$), Attribution X Actual Arousal X Sex ($p < .081$), and Attribution X Cognition of Arousal ($p < .071$; Table 7, p. 71).

Subjective report. The main effect for Attribution was far from significant. As reported previously, the Attribution X Cognition of Arousal interaction ($p < .017$) and Attribution X Actual Arousal X Sex interaction ($p < .038$) were significant (Table 8, p. 72). In addition to the previous analyses in terms of evidence for Hypotheses 1 and 2, these interactions can be analyzed in terms of simple effects for the Attribution factor and evidence for Hypothesis 3.

Concerning the Attribution X Cognition of Arousal interaction, the simple main effect for Attribution was significant for subjects who were told they were Aroused ($p < .018$) but not for subjects who were given No Information or subjects who were told they were relatively Unaroused (Table 17, p. 83). Within the Aroused condition of Cognition of Arousal, Intrinsic attribution subjects averaged higher Subjective Report of anxiety than subjects who were told their arousal was due to the drug injection; this is as predicted by Hypothesis 3. There were no simple interactions of Attribution with other factors to qualify this relationship within the Aroused condition.

Hypothesis 3 also predicted that the Intrinsic Attribution condition would produce greater Subjective Report than the Extrinsic condition for subjects in the Epinephrine condition but not for No Epinephrine subjects. Evidence for this part of the hypothesis was provided by the significant three-factor Attribution X Actual Arousal X Sex effect (Table 9, p. 73). The simple main effect for Attribution was not significant within the

Epinephrine or No Epinephrine conditions or for Females or Males. However, there was a significant Attribution X Actual Arousal interaction for Males ($p < .006$) but not for Females, as described earlier in connection with Actual Arousal. As can be seen in Table 9, the Intrinsic Attribution condition produced greater Subjective Report of anxiety than the Extrinsic Attribution for the average Male subject in the Epinephrine condition ($p < .010$) but not in the No Epinephrine condition. There was no interaction of Attribution with Cognition of Arousal for Males in the Epinephrine group to complicate this significant effect. Thus, Hypothesis 3 was supported, but only for Males for Actual Arousal.

Observed behavior. The main effect for Attribution was not significant for this dependent measure and neither were any interactions involving Attribution (Table 10, p. 75).

Tendency to avoid. The overall chi-square test for Attribution condition and Return versus Avoid was not significant and neither were the chi-square tests within conditions of the other factors, with one exception (Tables 19a and 19b). Attribution was significantly associated with Tendency to Avoid within the No Epinephrine condition. This was not expected, and neither was the direction; a higher proportion of Extrinsic subjects than of Intrinsic subjects were in the Avoid category.

Post-manipulation autonomic measures. Attribution was involved in one significant effect for adjusted Post-Manipulation measures. The Attribution X Actual Arousal X Sex interaction was significant for adjusted

Table 19a

Two X Two Contingency Table and Chi-Square for Attribution and Tendency to Avoid

| | Avoid | Return |
|---|---|---|
| Extrinsic | 20 | 28 |
| Intrinsic | 16 | 32 |
| $X^2 = .7111$ | | |

Table 19b

Two X Two Contingency Table and Chi-Square for Attribution and Tendency to Avoid Within the No Epinephrine Condition

| | Avoid | Return |
|---|---|---|
| Extrinsic | 12 | 12 |
| Intrinsic | 5 | 19 |
| $X^2 = 4.4629$ $p < .05$ | | |

Post-Manipulation Pulse Rate, as reported in connection with Actual Arousal (Table 16, p. 82). The simple effect for Attribution was not significant within the Epinephrine or No Epinephrine conditions or for Females or Males, but there was a significant Actual Arousal X Attribution interaction for Males ($p < .023$; Table 15, p. 81). The Attribution conditions did not differ significantly on adjusted Post-Manipulation Pulse Rate for Males in the No Epinephrine condition, but the Intrinsic condition produced significantly higher adjusted Post-Manipulation Pulse Rate for Males in the Epinephrine condition ($p < .014$).

## Relationships among Measures

A secondary concern in analyzing these data was the correlations among measures. This was of particular interest in view of the incongruities reported in some studies for autonomic, subjective, and behavioral responses. Pearson product-moment correlations were calculated between all pairings of the following measures: Subjective Report, Observed Behavior, Autonomic Report, Pre-Injection autonomic measures, Post-Injection autonomic measures, and Post-Manipulation autonomic measures (Table 20a). In addition, biserial correlations were performed for these measures and the Avoid and Return categories on Tendency to Avoid (Table 20b). The interpretation of these correlation coefficients must be tempered by the fact that different manipulations occurred in all the cells; however, the effects of the independent variables

## Table 20a

Product-Moment Correlation Coefficients for All Variables Analyzed by Analysis of Variance[a]

| | Observed Behavior | Autonomic Report | Pre-Injection Pulse Rate | Post-Injection Pulse Rate | Post-Manipulation Pulse Rate | Pre-Injection Systolic Blood Pressure |
|---|---|---|---|---|---|---|
| Subjective Report | .439 | .440 | .307 | .336 | .339 | .057 |
| Observed Behavior | | .278 | .332 | .387 | .369 | .020 |
| Autonomic Report | | | .141 | .229 | .223 | .059 |
| Pre-Injection Pulse Rate | | | | .883 | .888 | .173 |
| Post-Injection Pulse Rate | | | | | .926 | .144 |
| Post-Manipulation Pulse Rate | | | | | | .200 |

Table 20a (continued)

| | Post-Injection Systolic Blood Pressure | Post-Manipulation Systolic Blood Pressure | Pre-Injection Diastolic Blood Pressure | Post-Injection Diastolic Blood Pressure | Post-Manipulation Diastolic Blood Pressure |
|---|---|---|---|---|---|
| Subjective Report | .014 | .025 | -.109 | -.130 | -.144 |
| Observed Behavior | -.020 | -.032 | -.242 | -.232 | -.238 |
| Autonomic Report | .059 | .020 | -.002 | -.010 | -.031 |
| Pre-Injection Pulse Rate | .056 | .059 | .077 | .092 | .026 |
| Post-Injection Pulse Rate | .053 | .043 | -.008 | -.058 | -.133 |
| Post-Manipulation Pulse Rate | .099 | .113 | .017 | -.021 | -.097 |
| Pre-Injection Systolic Blood Pressure | .899 | .863 | .407 | .398 | .376 |
| Post-Injection Systolic Blood Pressure | | .938 | .405 | .411 | .437 |
| Post-Manipulation Systolic Blood Pressure | | | .437 | .475 | .452 |
| Pre-Injection Diastolic Blood Pressure | | | | .758 | .801 |
| Post-Injection Diastolic Blood Pressure | | | | | .878 |

[a] All correlation coefficients greater than .201 are significant at the .05 level, those greater than .262 are significant at the .01 level, for two-tailed tests.

Table 20b

Biserial Correlation Coefficients for Tendency to Avoid and All Variables Analyzed by Analysis of Variance

| | Coefficient |
|---|---|
| Subjective Report | .325[a] |
| Observed Behavior | .379[a] |
| Autonomic Report | .351[a] |
| Pre-Injection Pulse Rate | -.036 |
| Post-Injection Pulse Rate | .076 |
| Post-Manipulation Pulse Rate | .025 |
| Pre-Injection Systolic Blood Pressure | -.180 |
| Post-Injection Systolic Blood Pressure | -.152 |
| Post-Manipulation Systolic Blood Pressure | -.166 |
| Pre-Injection Diastolic Blood Pressure | .036 |
| Post-Injection Diastolic Blood Pressure | -.121 |
| Post-Manipulation Diastolic Blood Pressure | .163 |

[a] $p < .01$, two-tailed.

Table 20c

Partial Correlations for Subjective Report, Observed Behavior, and Autonomic Report with Significant Post-Injection and Post-Manipulation Autonomic Measures from Table 20b (Adjusted for Regression on Pre-Injection Levels)

| | Post-Injection Pulse Rate | Post-Manipulation Pulse Rate | Post-Injection Diastolic Blood Pressure | Post-Manipulation Diastolic Blood Pressure |
|---|---|---|---|---|
| Subjective Report | .147 | .153 | | |
| Observed Behavior | .212[a] | .171 | -.076 | -.076 |
| Autonomic Report | .225[a] | .215[a] | | |

[a] $p < .05$, two-tailed.

would contribute to the error term for the correlations; so we can regard the correlation coefficient for a pair of variables as some indication of the extent to which these variables were linearly related in this study.

Another caution is necessary in interpreting Tables 20a and 20b. Although performing intercorrelations is an acceptable way of describing linear relations within a set of data, the significance levels are relatively meaningless if many tests are performed, because of the number of tests itself and because the values of intercorrelations are mutually dependent (see Hays, 1963).

With these cautions in mind, several general observations can be made about the correlations. Except for the intercorrelations of the same autonomic measure at different times, the correlations are not high. The linear relationship of Subjective Report and the behavioral measures with Post-Injection and Post-Manipulation autonomic measures is nearly zero in some cases. In the other cases, the correlation is also high with the Pre-Injection measure. When these correlations were adjusted for the regression of each variable on the appropriate Pre-Injection autonomic measure by the method of partial correlation, the coefficients were substantially reduced (Table 20c).[1] Except for some of the Pre-Injection measures, which really have to do with the relations before the experiment

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[1] Correlations with Post-Manipulation levels were adjusted for regression on Pre-Injection rather than Post-Injection levels in order to include the total change in the autonomic measures after the experiment began.

began, the subjective and behavioral measures were more highly correlated with Autonomic Report than with the actual autonomic measures.

Although small, the correlations between Autonomic Report and Post-Injection Pulse Rate and Post-Manipulation Pulse Rate indicated some linear relationship. The correlation of Autonomic Report with Pre-Injection Pulse Rate was much less; consequently, the former two correlation coefficients were not appreciably changed by adjusting for the regression of the Autonomic Report and the Pulse Rate measure on Pre-Injection Pulse Rate (Table 20c). To determine whether the correlation between Autonomic Report and Post-Manipulation Pulse Rate was accounted for by the Post-Injection changes, the regression of Autonomic Report and Post-Manipulation Pulse Rate on Post-Injection Pulse Rate was eliminated; and the correlation practically disappeared ($r = .030$). Autonomic Report yielded coefficients of almost zero for correlations with the other autonomic measures.

In general, Subjective Report, Observed Behavior, Tendency to Avoid, and Autonomic Report show linear relations among themselves (and are more highly intercorrelated with each other than with Post-Injection or Post-Manipulation autonomic measures).[1]

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[1] These correlation coefficients are low, considering that a correlation coefficient of .4 indicates that the correlation with one variable accounts for only 16% of the variance on the other variable. Of course, in this study additional portions of the variance are accounted for by the treatments, in the case of most variables.

# DISCUSSION

## The Manipulations

Four main hypotheses were set forth in the Introduction. In general, the conditions for testing these hypotheses were met. Several points are worth noting in this connection.

### Actual arousal

A very small amount of epinephrine added to a solution of lidocaine hydrochloride proved sufficient to increase pulse rate an average of 6.263 beats per minute and decrease diastolic blood pressure an average of 5.155 millimeters of mercury relative to the level following injection of lidocaine hydrochloride without epinephrine.[1] This difference represents an actual average increase in pulse rate over the base level of about 6 beats per minute for the Epinephrine subjects as compared to no change for No Epinephrine. The actual diastolic blood pressure change is an average decrease from base level of about 6.5 millimeters of mercury compared to a fall of approximately 1.6 millimeters of mercury for No

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[1] Differences between Epinephrine and No Epinephrine cell means adjusted for regression of Post-Injection measure on Pre-Injection measure.

Epinephrine subjects.[1] Previous investigations of the autonomic effects of epinephrine included in dental anesthetics have yielded contradictory results (see Cheraskin & Prasertsuntarasai, 1958, and Persson, 1969, for reviews). The lack of consensus is partly due to lack of adjustments for initial level and to use of different local anesthetics, sites of injection (which affect absorption), epinephrine dosages, and intervals before measurement of the autonomic variables. For example, the effects of epinephrine depend on whether the alpha or beta receptors, or both, are activated; when both are fully activated by high dosages, alpha-receptor activity predominates in the cardiovascular system, but the beta receptors are sensitive at much lower concentrations and therefore predominate after the pharmacologically active concentration has dropped below the alpha-receptor threshold or at initially lower concentrations (Goodman & Gilman, 1970). Thus, the overall blood pressure response to epinephrine could be an increase, a decrease, or no change depending on dose, rate of absorption, and time of measurement.

Studies on the incidence of observed or reported side-effects (reviewed by Persson, 1969) are similarly inconsistent and difficult to interpret. In this case as well as when autonomic variables are measured after injection, many of the studies lack adequate controls or adequate statistical evaluation of the data. Practically all of the research on the

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[1] Unadjusted for regression of difference score on Pre-Injection level.

use of epinephrine with dental anesthetics has been performed to determine whether the minute amounts of epinephrine injected in dentistry could be harmful, especially for patients with cardiovascular disease. Therefore, small differences, such as found in the present study, have been regarded as inconsequential. Glover (1968) has gone even further in arguing that it is inconceivable that epinephrine doses as small as those in the present study could have any systemic effect unless accidentally injected intravenously. He estimated that 40 micrograms of epinephrine in a slowly injected dental anesthetic is absorbed over a 15 to 30 minute period and, due to elimination of some of the epinephrine before absorption, results in less than 1 microgram/minute entering the blood. Glover argues that this is much less than the amount secreted at rest (about 7 micrograms/minute) and therefore could have no systemic effect. However, it did have an effect in this study and similar amounts have had effects in other investigations. Goodman and Gilman (1970) note that some of the epinephrine (in concentrations of 1:50, 000 to 1:500, 000) included with local anesthetics may be absorbed systemically and occasionally lead to increased heart rate and palpitations along with other symptoms. In addition, although the difference was not tested statistically, Vernale (1960) found about the same effect on pulse rate in normotensive subjects as in the present study (about 6 beats per minute higher after 2 cubic centimeters of 2% lidocaine when it contained 20 micrograms of epinephrine than without epinephrine). With dosages 3-5 times higher than

those in the present study (5 cubic centimeters of 2% lidocaine with 1:50,000 epinephrine), Persson (1969) found significantly higher pulse rates and systolic blood pressures but no difference in diastolic blood pressures after injections containing epinephrine as compared to lidocaine injections containing no epinephrine. Several post-injection measurements were taken; at the point of greatest mean difference, pulse rate was 23.2 beats per minute higher and systolic blood pressure was 8.6 millimeters of mercury higher for the epinephrine injection. Subjects reported significantly more autonomic symptoms following the epinephrine injection, with the commonest symptom following epinephrine being palpitations. Frankenhaeuser and Järpe (1963) found similar effects with intravenous infusion of from 0.05 micrograms/kilogram/minute to 2.0 micrograms/kilogram/minute. Using Glover's estimates and 70 kilogram as the weight of an average male, the maximum dose in the present study (36 micrograms) would be comparable to a little over .01 micrograms/kilogram/minute for the average male or slightly more for the average female. Although not tested statistically, Frankenhaeuser and Järpe found increases in heart rate and systolic blood pressure and decreases in diastolic blood pressure relative to a placebo, even at the lowest epinephrine dosage. In addition, subjects exhibited tremor and reported palpitations and other autonomic symptoms even at the lowest dosage. Systemic effects of epinephrine at intravenous doses as low as .1 to .4 micrograms/kilogram/minute are well documented and include increases

in heart rate and systolic blood pressure and generally a decrease in diastolic blood pressure (Goodman & Gilman, 1970).

Although small relative to physiologically dangerous autonomic changes, the Post-Injection Pulse Rate and Diastolic Blood Pressure differences in this study were statistically significant. The Actual Arousal manipulations (anesthetic injections) were effective not merely in producing greater autonomic arousal in the Epinephrine as compared to No Epinephrine condition; in addition, for Females the Epinephrine injection also produced greater perceived autonomic arousal as measured by Autonomic Report.[1, 2]

The mechanism of the systemic effects of the minute amounts of epinephrine in the present study is unclear. Since the mechanism has no bearing on the evaluation of the hypotheses, only a few possibilities are advanced. One possible explanation is undetected intravenous injection of the anesthetic solutions in spite of aspiration before injection. This may have occurred in a few cases if the tip of the needle was within a blood vessel but against its wall or bone, but it is very improbable that this could have occurred in enough cases to account for the significant results. Alternatively, absorption may have been more rapid and metabolism and

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[1] Autonomic Report depended on other autonomic changes in addition to the cardiovascular effects measured. Epinephrine produces rather diffuse autonomic effects and each item on the Autonomic Report scale was reported by some subjects.

[2] Sex differences on this and other measures are dealt with in a later section.

elimination slower than that estimated by Glover (1968) for submucosal injections. The oral cavity is highly vascularized, and Pickering, Steinmeyer, and Luckhardt (1938) demonstrated greater hemodynamic responses following submucosal injections in the oral cavity in dogs than following subcutaneous injections of comparable amounts of local anesthetic with epinephrine.

The decrease in Post-Injection Diastolic Blood Pressure found in Epinephrine subjects was probably due to peripheral vasodilatation and decreased peripheral resistance occurring due to stimulation of beta receptors once the epinephrine had entered the circulation. The increased Post-Injection Pulse Rate could have been compensatory to the effect on blood pressure or due to direct cardiac effect of the epinephrine.[1] Continuous recording of autonomic measures would be necessary to evaluate these alternatives, but it was not feasible in this study. The present data did not indicate the time of onset of the autonomic responses nor did they necessarily indicate the maximum autonomic differences between Epinephrine and No Epinephrine groups after the injections.[2] However, the

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[1] Epinephrine in larger doses (intravenous infusion of .1 to .4 micrograms/kilogram/minute) increases stroke volume, cardiac output, and coronary blood flow (Goodman & Gilman, 1970). If these effects occurred in the present study, Autonomic Report of cardiac sensations probably reflected them in addition to, or rather than, increased pulse rate _per se_.

[2] In fact, the Epinephrine and No Epinephrine groups may have also differed significantly on Systolic Blood Pressure at some time other than one minute after the injection, although it is unlikely with the small dosages used.

Post-Injection autonomic measures did serve the purpose for which they were designed--they demonstrated the effectiveness of the Actual Arousal manipulation.

Cognition of degree of arousal

The nature of this manipulation was quite different from previous studies, but it was evidently quite effective as far as the Aroused condition was concerned. This demonstrated that it is possible to manipulate cognition of arousal by verbal communication. However, in this study it was easier to make people believe that they were Aroused than Unaroused. Although, as mentioned in the Method section, the Aroused manipulation could have been effective and still not have produced significantly greater Autonomic Report than No Information (since the subject could believe he is aroused yet unable to detect the arousal), this is not true of the Unaroused manipulation. If the Unaroused manipulation were effective, Unaroused subjects would not be expected to report as much autonomic arousal as No Information subjects. Even if they perceived autonomic symptoms, Unaroused subjects on the average should have rated themselves lower on the scaled Autonomic Report items than No Information subjects if they believed that their arousal was low relative to other oral surgery patients.

There are several possible explanations. The No Information subjects averaged only 1.469 as compared to 3.281 for Aroused subjects on Autonomic Report. It is possible that the lack of a significant difference

between the Unaroused and No Information groups is due to the low scores of the No Information group; however, the difference was also non-significant within the Epinephrine condition ($F < 1$) where the No Information mean was somewhat higher than over all 24 cells (1.935). Another possibility is that the Unaroused subjects inferred that the experimenter was trying to be reassuring by telling them that they were less aroused than they actually were. The manipulation for the Unaroused group was worded so as to be as comparable as possible to the feedback in bogus heart rate studies on avoidance; in these studies, "heart rate" remains constant during slides appropriate to the subject's fear but increases to another type of slide. The intent of the manipulation in the present study was to communicate the idea of no increase in arousal in the potentially arousing situation and to compare this arousal to higher levels (other patients rather than due to other stimuli); however, another tactic might have been more effective. Since many subjects were probably aroused prior to the first pulse and blood pressure measurements, telling them that their arousal had actually decreased might have been more effective. Alternatively, the manipulation could have been improved by presenting it in a way which would substantiate its "validity," for instance, by having the patients overhear the experimenter giving the dental assistant bogus pulse rate and blood pressure values to enter in the patient's chart, in addition to the Unaroused information presented to the subject.

The Aroused manipulation was effective. Although of less practical

value, it did permit testing the hypotheses concerning the effects of Cognition of Arousal. Another interesting finding contradicts Valins' (1966) report that subjects who heard bogus heart rate increases in the study on attractiveness of pin-up girls reported less palpitations than control subjects. Although the difference may be partly due to the nature of the feedback used, it does not seem that manipulations of cognition of arousal necessarily detract attention from actual arousal since Aroused subjects in this study reported more autonomic arousal than No Information subjects.

## Attribution of arousal

The Attribution manipulation warrants further attention before discussion of evidence for the hypotheses. We will limit our present considerations to the attributional process rather than attributional consequences.

The effectiveness of the attribution manipulation in the Schachter and Singer (1962) study was assessed only by an open-ended question which was used to eliminate "self-informing" subjects from the analyses, rather than being analyzed itself. Ross et al. (1969) used open-ended questions and specific questions dealing only with the induced attribution, for fear of artifactually inducing the nondesired attribution through questions relating to it. Nisbett and Schachter (1966) assessed the attribution manipulation with a post-experimental interview and then placed cases into categories

which did not allow examination of the number of subjects who apparently expressed both intrinsic and extrinsic attributions. From these studies there is little evidence for or against an assumption made in most discussions of attribution of arousal. The assumption is that physiological arousal prompts a search for a cause for that arousal and that when an extrinsic attribution manipulation is followed by less subjective emotion or emotional behavior than an intrinsic attribution, it is because the extrinsic attribution satisfies the need for finding the cause of the arousal, thereby preventing the search for other alternatives (Ross et al., 1969; Schachter & Singer, 1962). This being the case, it is surprising that subjects have not been questioned directly about their actual attributions of their arousal more often.

In the present study, since the manipulation check for the Attribution factor was included after the dependent measures in the questionnaire, subjects in both Attribution conditions could be asked about a variety of possible attributions. Out of the 66 subjects who expressed some perception of arousal on the Autonomic Report measure, 34 attributed their arousal to more than one cause.[1] And 13 of 19 subjects who attributed their arousal partly to the drug also reported anticipation of dental work as a cause. These data do not support the notion that possessing an extrinsic attribution for one's arousal precludes the simultaneous

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[1] Three subjects answered that none of the causes listed had contributed to their arousal, so the number that reported only one cause is 29.

acceptance of an intrinsic attribution. In this study subjects generally reported the intrinsic (anticipation of dental work) attribution or the extrinsic attribution (drug) and the intrinsic attribution.[1] Kelley's discussion of multiple plausible causes in relation to attribution of causality for another's behavior (1972a) is quite relevant. According to Kelley, attribution to one cause, for instance, something internal to the person, is relatively straightforward if that is the only plausible cause present. However, with the addition of another plausible cause, for instance, something external to the person, the presence of the other cause is less certain. The presence of the external cause makes the inference about the internal cause ambiguous rather than necessarily precluding it as a possibility. Kelley (1972b) extends this principle to attributions of one's own behavior, specifically to intrinsic and extrinsic attributions. The generalization to intrinsic and extrinsic attributions of one's own autonomic behavior when there are multiple plausible causes, as in all the attribution of arousal research, is direct and seems more reasonable than an "either-or" assumption.

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[1] Although not manipulated, attributions to answering the questionnaire and pain from the shot are technically extrinsic attributions; however, they are not included as such in this discussion. Only two subjects attributed any arousal to answering the questions. Conversations with many of the subjects revealed that they interpreted the pain question as an inquiry as to whether the shot had been painful or whether they had experienced arousal due to fear of pain from the shot. In addition, this attribution may not have occurred to the subjects until they read the question, whereas the drug attribution was salient from the time the information was given. For these reasons, the discussion of the extrinsic attribution is limited to the drug attribution.

This principle would also explain another effect, the limited effectiveness of the Extrinsic Attribution manipulation. Since the forthcoming dental work was a salient fact for Extrinsic as well as Intrinsic Attribution subjects, the presence of two plausible explanations limited the plausibility of the drug manipulation for the Extrinsic subjects. This finding is particularly interesting in light of the limitations on the plausibility of attributions found in previous studies (Nisbett & Schachter, 1966; Rosen et al., 1970; Schachter & Singer, 1962), as discussed in the Introduction.

Previous studies generally had difficulty in inducing misattributions, but in the present study there was difficulty in inducing the drug attribution even for Epinephrine subjects for whom it was true. For experimental purposes the salience of the drug manipulation could probably have been increased, for instance, by having a stooge pose as a particular dental student's previous patient and describe his autonomic reactions to the drug to the actual patient--a procedure which, however, would not be feasible in everyday dental practice. The simultaneous plausibility of the intrinsic attribution probably could not be further reduced in this context because anticipation of dental work is a particularly salient stimulus, especially for persons who have a history of fear of the dentist (as Rosen's subjects had a history of fear of snakes). However, in any situation in which we desire to inhibit an attribution having harmful subjective or behavioral effects, that attribution is likely to be quite salient.

Another fact which probably limited the effectiveness of the Attribution manipulation was that the onset of autonomic arousal could not be controlled. Subjects who were already aroused when the experiment began were unlikely to attribute much of their arousal to the drug. This could also account for the failure to induce a drug attribution in subjects expecting a strong shock in the Nisbett and Schachter (1966) study; in this study threat of shock was introduced well before the drug (placebo) was administered. The opposite problem probably accounted for Schachter's self-informing subjects; onset of symptoms may have occurred too precipitously to allow serious consideration of the intrinsic attribution. Ross et al. (1969) made a point of introducing noise and threat of shock practically simultaneously; but such simultaneity would not be possible in the therapeutic applications which they and Valins and Nisbett (1972) suggest for attributions of arousal.

It is possible but not probable that further limitations in the Attribution manipulation in the present study occurred due to telling the subjects in the preliminary instructions that many people are nervous in this situation. This was considered necessary to encourage honest answers on the questionnaire and was probably not a novel idea for the patients. The present study also differed from previous studies in that Intrinsic subjects were not misinformed as to the side effects of the drug; rather, they were simply told nothing about the drug. The previous attribution of arousal studies discussed herein used misattribution for the intrinsic

attribution condition to control for any effects on attention to autonomic activity due to the extrinsic manipulation. Since all subjects' attention was directed to their autonomic activity by repeated pulse and blood pressure measurements, misinforming the Intrinsic subjects was not deemed necessary in the present study, nor desirable since Ross et al. (1969) found that 80% of their misinformed subjects actually reported one or more of the supposedly irrelevant symptoms; this would inject another attributional process into an already complicated situation.

## The Major Findings

### Subjective report

Actual arousal X attribution X sex interaction. As predicted in Hypothesis 1, higher actual arousal produced greater report of subjective anxiety only for subjects for whom no extrinsic explanation was available for the arousal. However, this was true only for Males. The related prediction, in Hypothesis 3, that the Intrinsic Attribution condition would produce greater Subjective Report of anxiety than the Extrinsic manipulation only for subjects who had some arousal to attribute, due to the Epinephrine injection or the Aroused manipulation, was also supported only for Males when the Actual Arousal X Attribution interaction was considered.

These findings were somewhat surprising since only Females reported greater autonomic arousal in the Epinephrine than in the No Epinephrine condition. As an explanation of the presence of the Actual

Arousal X Attribution interaction and predicted effects for Males in the absence of a significant difference on Autonomic Report, we might conclude that the actual level of arousal (which was greater in the Epinephrine than in the No Epinephrine condition for Males) was of greater importance than the perceived level of arousal. However, the correlational data argue against this conclusion. After adjustment for regression on baseline levels, Subjective Report of anxiety was not correlated with Post-Injection autonomic measures, whereas Subjective Report of anxiety and Autonomic Report were linearly related. It is possible that the Males' sensitivity for internal autonomic changes was lower but equally possible that they experienced the same amount of arousal as Females but had different criteria for choosing a number on the Autonomic Report scale. In general, just as many Males as Females reported some autonomic arousal (31 Males and 35 Females over all cells; 21 Females and 19 Males in the Epinephrine cells); the difference was in the amount of arousal reported in the Epinephrine cells (Table 5, p. 67).

The original plan for the present experiment was to use subjects all of one sex, preferably females because of the common supposition that women in our society are more open than men in expressing their feelings, especially to another female (the experimenter). There are some indications that Males may have been downgrading their reactions. Males scored significantly lower on Subjective Report and were rated significantly lower on Observed Behavior than Females. But on the Tendency to

Avoid measure, which most subjects approached as though they might actually be asked to return for the experiment, although a few more Males were in the Avoid category, the association between Sex and Tendency to Return was not significant ($X^2 = 1.6000$). Of course, it is impossible to assess the comparability of the same score for Females and Males, or for any two groups. And Females may just have been more threatened by this situation for some reason.[1]

We are left with the unresolvable alternatives that Males may have experienced more autonomic arousal than they reported or that the predictions for Actual Arousal and Attribution in Hypotheses 1 and 3 were confirmed for Males in spite of the fact that the Epinephrine and No Epinephrine Male subjects differed only on Post-Injection Pulse Rate and Diastolic Blood Pressure and not on their perception of autonomic arousal. The results probably would have been more clear-cut if it had been possible to use larger doses of epinephrine.

The other side of this complexity is the absence of the predicted effects for Females in spite of their higher pulse rate, lower diastolic blood pressure, and greater Autonomic Report in the Epinephrine than in the No Epinephrine condition. There is no obvious explanation. As can be seen in Table 9 (p. 73), for Females both Epinephrine groups were

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[1] The possibility that the raters may have expected that Females would be more anxious cannot be dismissed as a possible explanation for their higher average on Observed Behavior.

higher (although not significantly) on Subjective Report of anxiety than the No Epinephrine groups. Moreover, the Females' Subjective Report showed less manipulability than the Males' Subjective Report. The means for Males conform closely to the hypothesis that both autonomic arousal and an appropriate (intrinsic) attribution for that arousal are necessary for Subjective Report of anxiety.[1] The induction of both of these factors (Epinephrine-Intrinsic condition) resulted in Subjective Report on the "tense," "nervous," "jittery" end of the scale. In the absence of either one, Subjective Report was on the "relaxed," "calm," "at ease" end of the scale. In contrast, the means for Females show neither as great an effect for the presence of both factors nor any substantial reduction due to less arousal (No Epinephrine-Intrinsic condition) or an alternative explanation for the arousal (Epinephrine-Extrinsic condition). In fact, the Epinephrine-Extrinsic mean is higher than the Epinephrine-Intrinsic mean. None of the means for Females are on the "relaxed," "calm," "at ease" end of the Subjective Report scale. This differential response

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[1] The term "necessary" is used very loosely here in connection with autonomic arousal. Strictly speaking, it implies a comparison of arousal with no arousal. Although there was, on the average, very little change from Pre-Injection measures (baseline) for the No Epinephrine subjects, we cannot conclude from these measures that there was no increase of sympathetic activity of any type for these subjects. In addition, most of the subjects were probably somewhat aroused on the Pre-Injection measures, as compared to their everyday baseline on those variables. However, the term "necessary" is commonly used in discussing the difference between a low and a higher state of arousal and will expedite the present discussion.

of Female and Male subjects to the interactions of Actual Arousal and Attribution conditions is interesting but inexplicable with the present data. Explanations in terms of differential effects of a female experimenter, overall Sex differences on Subjective Report, or general nonmanipulability of Female subjects would be convincing only if we could assume that these differences could affect the Actual Arousal X Attribution interaction but not the interaction for Cognition of Arousal X Attribution, which was significant and supported the predictions of Hypotheses 2 and 3 for both Females and Males. Likewise, there was a greater difference in the number of drug attributions in the Extrinsic as compared to the Intrinsic condition for Males (9 drug attributions in Extrinsic condition, 1 in Intrinsic) than for Females (6 drug attributions in Extrinsic condition, 3 in Intrinsic).[1] But if the Sex difference was due to attributional differences alone, it would be expected to affect the Cognition of Arousal X Attribution interaction as well, which it did not. It is most likely that the Sex differences appeared due to a combination of conditions, some of which were specific to the Actual Arousal X Attribution interaction. In addition, although the overall Sex differences in level of Subjective Report (and Observed Behavior) might well generalize to other similar studies, females and males are similar enough in their reactions to other variables

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[1] Considering the limited acceptance of the Extrinsic manipulation, the fact that Attribution of arousal produced significant effects under any conditions is surprising. This is discussed below under the Cognition of Arousal X Attribution interaction.

manipulated in psychological research that we would not be surprised to find significant actual arousal X attribution interactions for subjective report measures and support for the equivalents of Hypotheses 1 and 3 under slightly different conditions. The interaction with Sex found in the present study indicates both the pitfalls of including subjects of both sexes in the experimental design and the necessity for more psychological research which does so.

The Actual Arousal condition was included in this study as a test of Schachter's (1966; Schachter & Singer, 1962) hypothesis that physiological arousal is necessary for subjective emotion and emotional behavior. Support for this hypothesis and for Schachter's hypothesis that an appropriate attribution for that arousal is also a necessary condition was provided by the Subjective Report data from Male subjects. However, Schachter assumes that perception of the arousal acts as a cognitive cue and prompts the search for the attribution; these data do not support that assumption since Male Epinephrine subjects did not report perceiving significantly more autonomic arousal than Male No Epinephrine subjects. However, the Male subjects may not have reported all the autonomic arousal which they perceived. Since the process is assumed to be cognitive, and perception of arousal is a special case of cognition of arousal, the next section is relevant to this question.

Cognition of arousal X attribution. Significant attribution of arousal effects on Subjective Report were found for Females as well as Males within the Aroused condition of Cognition of Arousal. As predicted in Hypothesis 3, Intrinsic Attribution subjects reported more subjective anxiety than Extrinsic subjects when they were told that they were Aroused but not when they were told that they were Unaroused or given No Information. In addition, the Aroused condition produced greater Subjective Report of anxiety than the No Information condition only for subjects who were not provided with an extrinsic explanation for their arousal, as predicted in Hypothesis 2. Hypothesis 2 also predicted that subjects who were told that they were Unaroused would report significantly less subjective anxiety than subjects given No Information in the Intrinsic Attribution condition; this prediction was not supported, probably due to lack of acceptance of the Unaroused manipulation.

The effects for Attribution conditions and the Aroused and No

Information groups (Table 17, p. 83) parallel the interaction for Attribution and Actual Arousal for Males, although the means are higher in the former case due to inclusion of Females. The significant effects for Attribution within the Epinephrine and Aroused conditions, while expected on the basis of theoretical considerations, are surprising since so few Extrinsic Attribution subjects expressed the extrinsic (drug) attribution when asked for their opinion concerning the cause of their arousal.[1] Although many Extrinsic subjects did not report any attribution to the drug on their questionnaire, they all heard the experimenter explain the possible side-effects of the drug as including palpitations and tremor, and almost 90% of those Extrinsic subjects who reported some arousal reported palpitations and/or tremor. Therefore the drug attribution would seem to have been at least plausible for the Extrinsic subjects, whereas it probably did not even occur to most Intrinsic subjects.

Returning to Kelley's (1972a, 1972b) discussion of multiple plausible causes, it is probable that, in addition to those Extrinsic subjects who reported drug attributions, many other Extrinsic subjects became less confident of the attribution of their arousal to the dental situation after being made aware of another possible cause. From the significant interactions and simple effects for Attribution, it is apparent that the addition of another plausible cause for arousal was reassuring for aroused

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[1] Without the additional information from the manipulation check, it would have appeared from the results on Subjective Report that the Extrinsic subjects had accepted the manipulation wholeheartedly.

(Epinephrine or Aroused) subjects in contrast to the relative certitude that their arousal was produced by anticipation of dental work. This effect did not depend on the drug attribution being the sole attribution for the Extrinsic subjects nor on every Extrinsic subject's accepting the drug attribution for himself. Such effects were not found in the present study even though Attribution did affect Subjective Report as predicted. Since a small amount of ambiguity apparently affects the subject's inference about his state, larger differences would probably have been obtained within the Actual Arousal X Attribution and Cognition of Arousal X Attribution interactions if the drug attribution had been made more salient. In general, the hypotheses for attributions of arousal made by Nisbett and Schachter (1966), Ross et al. (1969), and Schachter and Singer (1962; Schachter, 1966) were supported by this experiment.

Valins' (1966; Valins & Ray, 1967) hypothesis of the comparability of direct manipulations of cognitions of degree of arousal to manipulations of actual arousal was also supported for Subjective Report. The fact that the Unaroused manipulation did not affect Subjective Report (when compared to No Information) is not evidence against his hypothesis since subjects evidently did not believe the manipulation. One might expect that manipulations of cognition of degree of arousal would be more effective when they are consonant with actual autonomic activity; however, this does not seem to be so within the range of arousal manipulated in the present study. Although the presence of significant effects for the Unaroused

condition as compared to No Information would have permitted a better test of this possibility, the present data allow us to conclude that the effects of Actual Arousal and Cognition of Arousal are additive rather than multiplicative, in the present study at least. Actual Arousal and Cognition of Arousal did not interact significantly, even within the Intrinsic Attribution condition, indicating that the Cognition of Arousal manipulation was effective regardless of the actual degree of arousal induced.

The significant effects for Cognition of Arousal and the fact that the effects for Aroused and No Information parallel those for Male Epinephrine subjects and Male No Epinephrine subjects in their interactions with Attribution lends credence to the assumption that autonomic activity functions as a cognitive cue.

We can conclude from these data on Subjective Report that both an intrinsic attribution _and either_ actual arousal _or_ information that one is aroused are necessary for report of subjective anxiety. Whereas the Schachter and Singer study provided evidence for this multiplicative effect for arousal and attribution only for the behavioral measures, the present investigation produced evidence only for Subjective Report.

## Behavioral measures

In general, the independent variables did not produce significant differences on behavioral measures in spite of the fact that the hypotheses were generally supported for Subjective Report. In light of the common assumption that one's behavior in a threatening situation depends largely

on one's subjective feeling, this discrepancy is quite interesting.

Nisbett and Valins (1972) advance the notion of hypothesis confirmation in connection with inferences about autonomic arousal. They suggest that much of a subject's behavior when he holds the cognition that he is aroused may be explained as attempts to validate inferences about that arousal. They suggest that such behavioral attempts at validation may precede a change in attitude, and they explain the significant behavioral effects in the absence of significant subjective report effects found by Valins and Ray (1967) on this basis. In the present study, the nature of the behavioral measures was such that they probably did not reflect attempts to validate inferences about the cognized arousal. Even for subjects who assumed that they would be asked to return to be in the experiment on the basis of their answer on the Tendency to Avoid measure, the experiment was understood to be planned well in the future.

The absence of significant effects on the behavioral measures used, in the presence of significant effects on the subjective measure, has a logical explanation. As pointed out in the Introduction, autonomic, subjective, and behavioral responses differ in their functions and in their control by external contingencies. We do not always express our feelings in our behavior; our behavior is partly dependent on our subjective feeling but also dependent on other considerations, including the expected consequences of that behavior. In the present investigation, although part of the variance on the behavioral measures was accounted for by their

linear relation with Subjective Report, the subjective and behavioral measures diverged in their relationship to the independent variables.

Nervous behavior, such as restlessness, is generally not reinforced by other people, especially not by complete strangers. And the dental situation in which the patient is expected to sit still and be cooperative would tend to further inhibit any behavioral disturbances which the patient was able to control, thereby reducing the effects of the manipulations on Observed Behavior. More direct evidence for the effect of motivations which are unrelated to the inferences about cognized arousal (due to Actual Arousal or Cognition of Arousal) and its source was found on the Tendency to Avoid measure. Subjects were asked to predict, on the basis of their own reactions, whether patients would return to be in an experiment similar to the experience of having a tooth extracted. The assumption that an answer in the negative reflected avoidance was substantiated by the fact that 34 out of 36 subjects in the Avoid category explained their response in terms of the aversiveness of the situation. On the other hand, an answer in the affirmative did not necessarily reflect a lack of subjective anxiety. Sixty subjects answered in the affirmative; they were asked what monetary inducement would be necessary and their reasons. Although many subjects would not make a monetary estimate or made one only after excessive prompting, the comments on this question were quite informative. Many subjects said that they personally would return for nothing, or transportation costs, or the pay they would lose on

their job, etc. Except for five subjects who said that an inducement would be necessary to compensate the subjects for pain or fear, their concern in predicting that patients would return and in estimating the inducement necessary was not with whether the situation did or did not have aversive effects; rather, they felt that subjects would return out of gratitude for inexpensive dental work or to "help out" the experimenter, the dental school, or future patients who might benefit from the research. Thus, the Tendency to Avoid measure was influenced by more than just the subject's inferences based on his cognized arousal and its apparent source.

In the present study, Observed Behavior really had no consequences relevant to the intrinsic attribution; that is, the subject who inferred "I am aroused because I am about to have a tooth pulled" really could do nothing about it by the time his behavior was observed by the dental student; therefore this inference had little effect on Observed Behavior. On the other hand, having made that inference he could decide on the Tendency to Avoid measure that neither he nor other subjects like him would return to a similar situation. However, the behavior of returning to be in an experiment had other, altruistic, consequences, and the predicted effects for cognized arousal and its attribution on Tendency to Avoid were not obtained.

A reasonable hypothesis seems to be that the effects of cognized arousal and attribution of that arousal on behavior are limited to the extent that there are expected behavioral consequences other than those

implied by the intrinsic attribution, i.e. "Removing myself from ________ will prevent my being aroused by ________." For instance, subjects in the Ross et al. (1969) study expected their attempts to solve the shock-avoidance puzzle (actually insoluble) to prevent their being shocked. No other consequences were foreseeable except for the possibility of losing a monetary reward, which the subjects estimated at $ .60, by not solving another puzzle presented at the same time. In this situation subjects who presumably made the inference "I am aroused by the threat of shock" worked largely on the shock-avoidance puzzle. What would be the effect of raising the monetary reward? Clearly, at some value the consequences of ignoring the reward puzzle would become too great and even subjects who inferred that they were aroused by threat of shock would confine their activity to the reward puzzle.

Similarly, the persistence of appropriate emotional behavior in Hohmann's (1966) paraplegic subjects (who had reduced autonomic sensations) can be explained on the basis of the external consequences. Although these subjects reported reduced subjective emotion as compared to before their injury, they reported that they avoided realistic dangers, and also that they "acted" emotional at times so that people would not think them odd.

It follows that behaviors such as phobias, which are by definition not tightly controlled by their external consequences, would be more reduced by a reduction in cognized arousal or the induction of an extrinsic

attribution for arousal than an appropriate avoidance behavior would be. Valins and Ray (1967) demonstrated that subjects with snake phobias exhibited greater approach behavior toward snakes after being induced to believe that they were not aroused by snakes. Such manipulations should be effective unless the phobia depends in addition on some underlying psychodynamic process or some consequence of the phobic behavior such as increased attention and sympathy from others.

So manipulations of cognized arousal and attribution of that arousal may have therapeutic effects for some behaviors as suggested by Valins and Ray (1967), Valins and Nisbett (1972), and Ross et al. (1969). These factors may be contributing causes for all behavior under stress, but to the extent that behaviors have other sufficient causes in the form of motivations to achieve or to avoid certain consequences, the effect of these factors will be reduced. In some situations manipulations of external contingencies will have greater effect, even on avoidance behavior, than manipulations of level of cognized arousal or attribution of arousal. A number of studies have shown that lower cognized arousal or an extrinsic attribution, as compared to high arousal and an intrinsic attribution for arousal, reduces emotional behavior, but they have demonstrated this in situations with no additional consequences for that behavioral measure. In light of the results in the present study, we cannot conclude from previous studies that the same reduction will occur when the behavior in question has other desirable consequences; nor can we conclude that high

cognized arousal and an intrinsic attribution necessarily produce emotional behavior if there are undesirable consequences for emotional behavior in that situation. Behavioral effects obtained on one type of measure cannot be generalized to other types of behavior if they differ in their consequences. Thus subjective anxiety may differ significantly due to manipulations of cognized arousal and its attribution without comparable effects in overt behavior. The Schachter and Singer (1962) and Valins and Ray (1967) studies show that the converse is also true, at least for manipulations of cognized arousal (when an intrinsic attribution for arousal has been induced). These discrepancies further demonstrate the fallacy of the common practice of extending conclusions from behavioral data to hypotheses about subjective feelings, and vice versa.

### Post-manipulation autonomic measures

Post-Manipulation Pulse Rate, Systolic Blood Pressure, and Diastolic Blood Pressure (each adjusted for Post-Injection level on the same measure) did not differ significantly across Cognition of Arousal conditions, supporting Hypothesis 4. Since Cognition of Arousal did have significant effects in this study, the explanation of the effects of induced cognitions in purely cognitive terms as proposed by Valins (1966) seems appropriate, at least for the present study. The data from this study do not bear on the possibility that actual changes in arousal do occur when continuous bogus heart rate feedback is used. But they do show that significant effects for induced cognitions of arousal can occur in the absence

of corresponding changes on the autonomic variables to which the Cognition of Arousal manipulations referred.

Although the Cognition of Arousal manipulation did not produce significant differences on the adjusted Post-Manipulation measures, the other factors did. Actual Arousal interacted with Sex in the case of diastolic blood pressure and with Sex and Attribution in the case of pulse rate (Table 14, p. 81); in both cases, arousal was significantly greater (lower diastolic blood pressure, higher pulse rate) for Males who had received Epinephrine than for Males who had received No Epinephrine. In addition, on the pulse rate measure, Actual Arousal and Attribution interacted for Males. Since the regression on the Post-Injection level of these measures was eliminated from the adjusted scores by ANACOVA, we can conclude that induction of different Actual Arousal levels had effects on further autonomic changes that were not linearly related to the actual values of the measures at the time of induction. The most logical explanation is that the Post-Manipulation differences for Males were mediated by another variable, presumably perception of autonomic arousal or subjective anxiety, or both. Mediation by subjective anxiety is most likely since the effect was not obtained for Females, who reported significantly more autonomic arousal in the Epinephrine than the No Epinephrine condition, and since the effects on the adjusted Post-Manipulation measures are consistent with the Actual Arousal X Attribution interaction for Males on Subjective Report. Since this effect did not occur for Cognition

of Arousal, it might depend on subjective anxiety interacting with the already high Post-Injection arousal in the Epinephrine condition.[1] However, this effect should not be overemphasized considering our limited understanding of the Actual Arousal X Sex interaction for Autonomic Report and the Actual Arousal X Attribution X Sex interaction for Subjective Report.

Males who received an epinephrine injection and did not have an extrinsic attribution for their arousal had significantly higher Post-Manipulation Pulse Rate than Males who received epinephrine but were provided with an extrinsic attribution for their arousal. Schachter and Singer (1962) hypothesized that, given some arousal, attributional effects on subjective report and behavior would occur even if the groups with different attributions for arousal were equal on arousal. Previous research did not bear on this hypothesis. In the present study the Attribution of Arousal condition was associated with differences in pulse rate for Males, but this does not rule out the possibility that attributional effects could occur in the absence of differences on arousal.

## Conclusions

With the exception of Females in the Actual Arousal X Attribution interaction and the Unaroused group in the Cognition of Arousal X

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[1] Although this difference in Post-Injection arousal was removed by the ANACOVA for analysis of the Post-Manipulation measures, it was present in reality when the effect occurred.

Attribution interaction, the four specific hypotheses advanced for this investigation were supported for Subjective Report. Considered in the context of the research reviewed in the Introduction, this investigation conducted on a heterogeneous sample of people faced with a truly stressful situation supports a cognitive interpretation of the effects of autonomic arousal on subjective report of anxiety. Specifically, the data support the hypothesis that both cognized arousal and an intrinsic attribution for that arousal are necessary for subjective report of anxiety. The cognized arousal may arise from actual arousal or information that one is aroused; in either case, it produces subjective anxiety if only an intrinsic attribution for arousal is available.

Nisbett and Valins (1972) have extended Bem's (1967) proposition that attitudes are inferred from behavior to include the inference of feelings from autonomic activity. However, there are important differences between overt behavior and autonomic behavior. As Nisbett and Valins point out, attitudinal inferences are made only if we perceive ourselves to have been responsible for the behavior, in other words, if the behavior was voluntary. But we generally do not perceive our autonomic behavior to have been voluntary. It is more likely that we infer subjective anxiety on the basis of autonomic cues because they have been associated in the past in stressful situations which produced diffuse and perceptible arousal. As a result of this repeated association, neither the situation nor the arousal are sufficient to produce the label of subjective anxiety; both are

necessary. Although an appropriate situation is not sufficient for the report of subjective anxiety, the controlling factors for overt behavior are different. The contingencies of the present situation affect overt behavior even in the absence of autonomic cues. And external contingencies may override the effect of high cognized arousal and an intrinsic attribution on voluntary behavior.

# APPENDIX A

# ORAL INSTRUCTIONS AND CONSENT FORM

## ORAL INSTRUCTIONS

(Pulse and Blood Pressure Measurement)

Instructions: We know that going to the dentist is often unpleasant and that it makes some people nervous. So we are doing a study to try to find out how to make dental patients feel more comfortable. You can help us by answering some questions in a few minutes. Your name will not be on your answer sheet, so no one will know which answers are yours when we look at them later. Please read this consent form and sign it if you agree with it.

(Administration of Consent Form)

(Injection)

(Pulse and Blood Pressure Measurement)

(Attribution and Cognition of Arousal Manipulations)

(Pulse and Blood Pressure Measurement)

Instructions: These questions are about how you feel. There are no right or wrong answers; we are just interested in your opinion. As I said before, we know that many patients find this situation unpleasant. Although it is sometimes hard to admit how you feel, you will be helping us only if you are completely truthful. Let me remind you that no one will know

which answer sheet is yours anyway.

(Administration of Questionnaire)

# CONSENT FOR CLINICAL RESEARCH

The purpose of this project is to understand the patient's response to dental treatment. It will involve no change from standard procedures in your dental treatment. You will simply be asked to fill out a questionnaire about your feelings; all of your answers will be kept confidential.

I agree to participate in this clinical research project at the School of Dentistry, University of North Carolina. And I agree to answer the questions as honestly as I can.

Signed: ______________________________

Date: ____________________________

Witness: _________________________

# APPENDIX B

# PILOT STUDY

# REPORT OF AUTONOMIC AROUSAL FOLLOWING DENTAL INJECTIONS OF EPINEPHRINE

Ten subjects, all dental students or student assistants or hygienists, were paid $5.00 for receiving two inferior alveolar blocks. One injection of approximately 2.7 cubic centimeters of lidocaine hydrochloride and one of approximately 2.7 cubic centimeters of lidocaine hydrochloride with epinephrine (1:100,000) were given on the same side of the mouth. The injections were separated by at least two hours; five subjects received the injection containing epinephrine first and five received the injection without epinephrine first. To prevent bias due to expectations concerning the second injection, subjects were told that they might receive the same anesthetic twice. Subjects were not informed about the actual order of presentation until their second session was completed.

After each injection the subject was asked to rate his sensations from 0 to 3 for palpitations, tremor, increased heart rate, increased respiratory rate, and increased respiratory depth. After the second injection each subject was asked whether he thought he had received the same solution twice or two different anesthetic solutions, and, if different, which had produced the most autonomic arousal. The data from one subject had to be discarded; she admitted experiencing a tremor (following epinephrine) which was visible to the experimenter but did not report any symptoms on the questionnaire, and she asked if the experiment was designed to ascertain the subjects' fear of shots. Seven out of the

remaining nine subjects rated themselves as more aroused, that is, they reported more specific changes and/or greater magnitude of change, following epinephrine administration (_t_ test for paired observations, $p < .06$, one-tailed). These seven also judged the two solutions to be different with the epinephrine solution definitely producing greater feeling of arousal. Several subjects offered the spontaneous observation that one injection made them feel "nervous," "like an emotion," or "excited"; this was the anesthetic containing epinephrine in every case. One subject rated the no-epinephrine solution as more arousing (although she reported more arousal for the epinephrine solution than any other subject). And, finally, one subject reported no difference in the amount of arousal perceived following the two injections.

Total Autonomic Report Scores

| Subject | Epinephrine | No Epinephrine |
|---|---|---|
| 1* | 5 | 1 |
| 2 discarded | - | - |
| 3* | 3 | 1 |
| 4 | 6 | 12 |
| 5 | 2 | 2 |
| 6* | 5 | 2 |
| 7* | 2 | 0 |
| 8* | 5 | 0 |
| 9* | 4 | 0 |
| 10* | 5 | 0 |

*These subjects concluded from their physiological reactions (excluding numbness of the mouth) that they had been given two different types of anesthetic and identified the anesthetic containing epinephrine (known to them only as the first or second injection) as the most autonomically arousing.

# APPENDIX C

# SUBJECTS' QUESTIONNAIRE AND OBSERVERS' RATING ITEM

## SUBJECTS' QUESTIONNAIRE

On each line below, circle the number which comes closest to the way you feel right now. If you feel more like one word, mark a number nearer to that word. If you don't feel like either word, mark the "0" in the middle.

| | Very Much | Pretty Much | A Little | Neither | A Little | Pretty Much | Very Much | |
|---|---|---|---|---|---|---|---|---|
| 1. Tense | 3 | 2 | 1 | 0 | 1 | 2 | 3 | Relaxed |
| 2. Calm | 3 | 2 | 1 | 0 | 1 | 2 | 3 | Nervous |
| 3. Jittery | 3 | 2 | 1 | 0 | 1 | 2 | 3 | At ease |

We may do another study sometime. It would be very much like getting a tooth pulled, but we would use people whose teeth were in good shape. The study would not harm their good teeth, but there would be a shot and instruments put in the mouth like today. There would be about the same amount of pain as today. People would be paid to come back when their teeth are in good shape. Do you think they would choose to come back based on your own experience here? ______________________

If not, why not? ______________________________________________

If yes, what do you think would be a fair wage to pay them?
$________ per hour

Why that amount? ______________________________________________

Circle the number which is closest to the way you have felt since the first blood pressure measure.

| | Not at All | A Little | Pretty Much | Very Much |
|---|---|---|---|---|
| 1. Have you felt your heart pounding? | 0 | 1 | 2 | 3 |
| 2. Have you felt any tremor (shaking)? | 0 | 1 | 2 | 3 |
| 3. Have you felt your heart beating faster than normal? | 0 | 1 | 2 | 3 |
| 4. Have you felt yourself breathing deeper than usual? | 0 | 1 | 2 | 3 |
| 5. Have you felt yourself breathing faster than normal? | 0 | 1 | 2 | 3 |

If you noticed any of the above changes, what do you think caused them?

________________________________________________________

Sometimes it is hard to say what causes these kinds of physical changes. If you noticed any of the physical changes in the list on the last page, how much do you think each of the possible causes below applies to you? Circle a number on each line.

| | | Not at All | A Little | Pretty Much | Very Much |
|---|---|---|---|---|---|
| 1. | The dental work that's about to start | 0 | 1 | 2 | 3 |
| 2. | The drug | 0 | 1 | 2 | 3 |
| 3. | Answering so many questions | 0 | 1 | 2 | 3 |
| 4. | Pain from the shot | 0 | 1 | 2 | 3 |

## OBSERVERS' RATING ITEM

How nervous or anxious do you think your patient was in the period between the administration of the questionnaire and contact of the tooth with the instruments (do not consider tremor)?

| Not at All | A Little | Pretty Much | Very Much |
|---|---|---|---|
| 0 | 1 | 2 | 3 |

# APPENDIX D

# TABLES FOR PRELIMINARY ANALYSES

Table 21

Summary of Analysis of Variance: Severity of Dental Procedure As a Function of All Independent Variables

| Source | df | MS | F | p |
|---|---|---|---|---|
| Actual Arousal (A) | 1 | 1.500 | < 1 | |
| Attribution (B) | 1 | 2.042 | < 1 | |
| Cognition of Arousal (C) | 2 | 2.042 | < 1 | |
| Sex (D) | 1 | .167 | < 1 | |
| A X B | 1 | 2.667 | 1.263 | n. s. |
| A X C | 2 | .500 | < 1 | |
| A X D | 1 | .042 | < 1 | |
| B X C | 2 | 3.292 | 1.559 | n. s. |
| B X D | 1 | .667 | < 1 | |
| C X D | 2 | 3.292 | 1.559 | n. s. |
| A X B X C | 2 | 1.167 | < 1 | |
| A X B X D | 1 | 1.042 | < 1 | |
| A X C X D | 2 | 2.542 | 1.204 | n. s. |
| B X C X D | 2 | .292 | < 1 | |
| A X B X C X D | 2 | 4.292 | 2.033 | n. s. |
| Error | 72 | 2.111 | | |

Table 22a

Summary of Analysis of Variance Within Epinephrine Condition: Amount of Anesthetic (Lidocaine with Epinephrine) as a Function of Remaining Independent Variables

| Source | df | MS | F | p |
|---|---|---|---|---|
| Attribution (B) | 1 | .152 | 1.235 | n. s. |
| Cognition of Arousal (C) | 2 | .366 | 2.975 | .064 |
| Sex (D) | 1 | .097 | < 1 | |
| B X C | 2 | .032 | < 1 | |
| B X D | 1 | .047 | < 1 | |
| C X D | 2 | .840 | 6.830 | .003 |
| B X C X D | 2 | .202 | 1.647 | n. s. |
| Error[a] | 36 | .123 | | |

[a] Within-cells error from Epinephrine cells.

Table 22b

Summary of Analysis of Variance Within No Epinephrine Condition: Amount of Anesthetic (Lidocaine Without Epinephrine) as a Function of Remaining Independent Variables

| Source | df | MS | F | p |
|---|---|---|---|---|
| Attribution (B) | 1 | .005 | < 1 | |
| Cognition of Arousal (C) | 2 | .328 | 2.077 | n. s. |
| Sex (D) | 1 | .005 | < 1 | |
| B X C | 2 | .068 | < 1 | |
| B X D | 1 | .880 | 5.571 | .024 |
| C X D | 2 | .161 | 1.022 | n. s. |
| B X C X D | 2 | .380 | 2.407 | n. s. |
| Error[a] | 36 | .158 | | |

[a] Within-cells error from No Epinephrine cells.

### Table 23a

Amount of Anesthetic by Cognition of Arousal Within Epinephrine Condition

| Unaroused | No Information | Aroused |
|---|---|---|
| 1.306 | 1.531 | 1.244 |

### Table 23b

Amount of Anesthetic by Cognition of Arousal and Sex Within Epinephrine Condition

| | Female | Male |
|---|---|---|
| Unaroused | 1.325 | 1.288 |
| No Information | 1.250 | 1.813 |
| Aroused | 1.413 | 1.075 |

### Table 23c

Amount of Anesthetic by Attribution and Sex Within No Epinephrine Condition

| | Female | Male |
|---|---|---|
| Extrinsic | 1.083 | 1.333 |
| Intrinsic | 1.375 | 1.083 |

Table 24a

Summary of Analysis of Covariance Within Epinephrine Condition: Post-Injection Pulse Rate with Pre-Injection Pulse Rate as a Covariate as a Function of Remaining Independent Variables

| Source | df | MS | F | p |
|---|---|---|---|---|
| Attribution (B) | 1 | 5.922 | < 1 | |
| Cognition of Arousal (C) | 2 | 35.469 | < 1 | |
| Sex (D) | 1 | 21.793 | < 1 | |
| B X C | 2 | 16.471 | < 1 | |
| B X D | 1 | 2.848 | < 1 | |
| C X D | 2 | 145.547 | 3.169 | .054 |
| B X C X D | 2 | 72.121 | 1.570 | n. s. |
| Within-cells regression | 1 | 4592.367 | 99.980 | .001 |
| Error[a] | 35 | 45.933 | | |

[a] Within-cells error from Epinephrine cells.

Table 24b

Summary of Analysis of Covariance Within Epinephrine Condition: Post-Injection Pulse Rate with Pre-Injection Pulse Rate and Amount of Anesthetic as Covariates as a Function of Remaining Independent Variables

| Source | df | MS | F | p |
|---|---|---|---|---|
| Attribution (B) | 1 | 1.738 | < 1 | |
| Cognition of Arousal (C) | 2 | 38.377 | < 1 | |
| Sex (D) | 1 | 18.262 | < 1 | |
| B X C | 2 | 16.680 | < 1 | |
| B X D | 1 | 4.211 | < 1 | |
| C X D | 2 | 148.369 | 3.151 | .056 |
| B X C X D | 2 | 73.342 | 1.557 | n.s. |
| Within-cells regression | 2 | 2299.469 | 48.831 | .001 |
| Error[a] | 34 | 47.091 | | |

[a] Within-cells error from Epinephrine cells.

Table 25a

Summary of Analysis of Variance Within Epinephrine Condition: Effects of Remaining Independent Variables on Subjective Report

| Source | df | MS | F | p |
|---|---|---|---|---|
| Attribution (B) | 1 | 30.083 | 1.869 | n. s. |
| Cognition of Arousal (C) | 2 | 70.021 | 4.350 | .020 |
| Sex (D) | 1 | 40.333 | 2.506 | n. s. |
| B X C | 2 | 16.146 | 1.003 | n. s. |
| B X D | 1 | 85.333 | 5.301 | .027 |
| C X D | 2 | 76.896 | 4.777 | .014 |
| B X C X D | 2 | 13.271 | < 1 | |
| Error[a] | 36 | 16.098 | | |

[a] Within-cells error from Epinephrine cells.

## Table 25b

Summary of Analysis of Covariance Within Epinephrine Condition: Effects of Remaining Independent Variables on Subjective Report with Amount of Anesthetic as a Covariate

| Source | df | MS | F | p |
|---|---|---|---|---|
| Attribution (B) | 1 | 47.418 | 3.240 | .081 |
| Cognition of Arousal (C) | 2 | 56.058 | 3.830 | .031 |
| Sex (D) | 1 | 30.012 | 2.050 | n. s. |
| B X C | 2 | 18.757 | 1.281 | n. s. |
| B X D | 1 | 69.722 | 4.763 | .036 |
| C X D | 2 | 100.594 | 6.873 | .003 |
| B X C X D | 2 | 14.460 | $< 1$ | |
| Within-cells regression | 1 | 67.208 | 4.592 | .039 |
| Error[a] | 35 | 14.638 | | |

[a] Within-cells error from Epinephrine cells.

### Table 26a

Summary of Analysis of Variance: Pre-Injection Pulse Rate as a Function of All Independent Variables

| Source | df | MS | F | p |
|---|---|---|---|---|
| Actual Arousal (A) | 1 | 15.041 | < 1 | |
| Attribution (B) | 1 | 715.038 | 4.244 | .043 |
| Cognition of Arousal (C) | 2 | 202.538 | 1.202 | n.s. |
| Sex (D) | 1 | 3577.009 | 21.230 | .001 |
| A X B | 1 | 287.045 | 1.704 | n.s. |
| A X C | 2 | 5.543 | < 1 | |
| A X D | 1 | 15.041 | < 1 | |
| B X C | 2 | 849.525 | 5.042 | .009 |
| B X D | 1 | 117.043 | < 1 | |
| C X D | 2 | 762.525 | 4.526 | .014 |
| A X B X C | 2 | 363.540 | 2.158 | n.s. |
| A X B X D | 1 | 15.041 | < 1 | |
| A X C X D | 2 | 502.541 | 2.983 | .057 |
| B X C X D | 2 | 96.542 | < 1 | |
| A X B X C X D | 2 | 347.537 | 2.063 | n.s. |
| Error | 72 | 168.488 | | |

## Table 26b

## Summary of Analysis of Variance: Pre-Injection Systolic Blood Pressure as a Function of All Independent Variables

| Source | df | MS | F | p |
|---|---|---|---|---|
| Actual Arousal (A) | 1 | 126.043 | < 1 | |
| Attribution (B) | 1 | 126.043 | < 1 | |
| Cognition of Arousal (C) | 2 | 303.034 | 2.178 | n. s. |
| Sex (D) | 1 | 782.044 | 5.621 | .020 |
| A X B | 1 | 222.044 | 1.596 | n. s. |
| A X C | 2 | 163.042 | 1.172 | n. s. |
| A X D | 1 | 45.373 | < 1 | |
| B X C | 2 | 53.792 | < 1 | |
| B X D | 1 | 100.038 | < 1 | |
| C X D | 2 | 24.542 | < 1 | |
| A X B X C | 2 | 448.780 | 3.226 | .046 |
| A X B X D | 1 | 260.031 | 1.869 | n. s. |
| A X C X D | 2 | 78.876 | < 1 | |
| B X C X D | 2 | 351.289 | 2.525 | .087 |
| A X B X C X D | 2 | 66.292 | < 1 | |
| Error | 72 | 139.129 | | |

Table 26c

Summary of Analysis of Variance: Pre-Injection Diastolic Blood Pressure as a Function of All Independent Variables

| Source | df | MS | F | p |
|---|---|---|---|---|
| Actual Arousal (A) | 1 | 121.503 | < 1 | |
| Attribution (B) | 1 | 80.665 | < 1 | |
| Cognition of Arousal (C) | 2 | 349.125 | 2.834 | .065 |
| Sex (D) | 1 | 88.168 | < 1 | |
| A X B | 1 | 1.500 | < 1 | |
| A X C | 2 | 304.877 | 2.475 | .091 |
| A X D | 1 | 95.998 | < 1 | |
| B X C | 2 | 179.790 | 1.451 | n. s. |
| B X D | 1 | 73.501 | < 1 | |
| C X D | 2 | 98.038 | < 1 | |
| A X B X C | 2 | 49.876 | < 1 | |
| A X B X D | 1 | 23.998 | < 1 | |
| A X C X D | 2 | 6.124 | < 1 | |
| B X C X D | 2 | 423.873 | 3.441 | .037 |
| A X B X C X D | 2 | 208.624 | 1.693 | n. s. |
| Error | 72 | 123.183 | | |

Table 27a

Summary of Analysis of Covariance Within Epinephrine Condition: Post-Injection Pulse Rate with Pre-Injection Pulse Rate as a Covariate as a Function of Remaining Independent Variables

| Source | df | MS | F | p |
|---|---|---|---|---|
| Attribution (B) | 1 | 5.512 | < 1 | |
| Cognition of Arousal (C) | 2 | 35.363 | < 1 | |
| Sex (D) | 1 | 21.961 | < 1 | |
| B X C | 2 | 17.408 | < 1 | |
| B X D | 1 | 2.941 | < 1 | |
| C X D | 2 | 155.973 | 3.556 | .034 |
| B X C X D | 2 | 72.900 | 1.662 | n. s. |
| Within-cells regression[a] | 1 | 8289.574 | 188.978 | .001 |
| Error[b] | 71 | 43.865 | | |

[a] Pooled within-cells regression from Epinephrine and No Epinephrine cells.

[b] Within-cells error from Epinephrine and No Epinephrine cells.

Table 27b

Summary of Analysis of Covariance Within No Epinephrine Condition: Post-Injection Pulse Rate with Pre-Injection Pulse Rate as a Covariate as a Function of Remaining Independent Variables

| Source | df | MS | F | p |
|---|---|---|---|---|
| Attribution (B) | 1 | 75.500 | 1.721 | n. s. |
| Cognition of Arousal (C) | 2 | 4.215 | < 1 | |
| Sex (D) | 1 | .074 | < 1 | |
| B X C | 2 | 3.371 | < 1 | |
| B X D | 1 | 0.000 | < 1 | |
| C X D | 2 | 1.973 | < 1 | |
| B X C X D | 2 | 131.205 | 2.991 | .057 |
| Within-cells regression[a] | 1 | 8289.574 | 188.978 | .001 |
| Error[b] | 71 | 43.865 | | |

[a] Pooled within-cells regression from Epinephrine and No Epinephrine cells.

[b] Within-cells error from Epinephrine and No Epinephrine cells.

## Table 27c

Summary of Analysis of Covariance Within Epinephrine Condition: Post-Injection Systolic Blood Pressure with Pre-Injection Systolic Blood Pressure as a Covariate as a Function of All Remaining Independent Variables

| Source | df | MS | F | p |
|---|---|---|---|---|
| Attribution (B) | 1 | 10.590 | $< 1$ | |
| Cognition of Arousal (C) | 2 | 118.697 | 3.461 | .037 |
| Sex (D) | 1 | 18.344 | $< 1$ | |
| B X C | 2 | 20.033 | $< 1$ | |
| B X D | 1 | 1.656 | $< 1$ | |
| C X D | 2 | 26.189 | $< 1$ | |
| B X C X D | 2 | 39.971 | 1.165 | n.s. |
| Within-cells regression[a] | 1 | 9259.859 | 269.982 | .001 |
| Error[b] | 71 | 34.298 | | |

[a] Pooled within-cells regression from Epinephrine and No Epinephrine cells.

[b] Within-cells error from Epinephrine and No Epinephrine cells.

## Table 28a

Post-Injection Systolic Blood Pressure Adjusted for Pre-Injection Systolic Blood Pressure by Cognition of Arousal Within Epinephrine Condition

| Unaroused | No Information | Aroused |
|---|---|---|
| 119.557 | 123.336 | 123.038 |

## Table 28b

Post-Injection Pulse Rate Adjusted for Pre-Injection Pulse Rate by Cognition of Arousal and Sex Within Epinephrine Condition

| | Female | Male |
|---|---|---|
| Unaroused | 81.690 | 90.083 |
| No Information | 87.123 | 87.283 |
| Aroused | 86.550 | 82.109 |

## Table 28c

Post-Injection Pulse Rate Adjusted for Pre-Injection Pulse Rate by Attribution and Cognition of Arousal and Sex Within No Epinephrine Condition

| | Extrinsic | | Intrinsic | |
|---|---|---|---|---|
| | Female | Male | Female | Male |
| Unaroused | 74.482 | 80.389 | 83.030 | 78.429 |
| No Information | 82.550 | 76.256 | 77.816 | 83.829 |
| Arousal | 78.030 | 78.216 | 81.816 | 80.083 |

## Table 29a

### Summary of Analysis of Variance Within Epinephrine Condition: Effects of Remaining Independent Variables on Subjective Report

| Source | df | MS | F | p |
|---|---|---|---|---|
| Attribution (B) | 1 | 30.083 | 1.929 | n. s. |
| Cognition of Arousal (C) | 2 | 70.020 | 4.489 | .015 |
| Sex (D) | 1 | 40.333 | 2.586 | n. s. |
| B X C | 2 | 16.146 | 1.035 | n. s. |
| B X D | 1 | 85.333 | 5.471 | .022 |
| C X D | 2 | 76.895 | 4.930 | .010 |
| B X C X D | 2 | 13.271 | < 1 | |
| Error[a] | 72 | 15.599 | | |

[a] Within-cells error from Epinephrine and No Epinephrine cells.

Table 29b

Summary of Analysis of Covariance Within Epinephrine Condition: Effects of Remaining Independent Variables on Subjective Report with Post-Injection Pulse Rate, Systolic Blood Pressure, and Diastolic Blood Pressure as Covariates

| Source | df | MS | F | p |
|---|---|---|---|---|
| Attribution (B) | 1 | 7.171 | < 1 | |
| Cognition of Arousal (C) | 2 | 92.183 | 6.438 | .003 |
| Sex (D) | 1 | 25.469 | 1.779 | n. s. |
| B X C | 2 | 12.904 | < 1 | |
| B X D | 1 | 102.206 | 7.137 | .009 |
| C X D | 2 | 32.947 | 2.301 | n. s. |
| B X C X D | 2 | 12.432 | < 1 | |
| Within-cells regression[a] | 3 | 44.980 | 3.141 | .031 |
| Error[b] | 69 | 14.317 | | |

[a] Pooled within-cells regression from Epinephrine and No Epinephrine cells.

[b] Within-cells error from Epinephrine and No Epinephrine cells.

Table 30a

Summary of Analysis of Variance Within No Epinephrine Condition: Effects of Remaining Independent Variables on Subjective Report

| Source | df | MS | F | p |
|---|---|---|---|---|
| Attribution (B) | 1 | 27.000 | 1.731 | n. s. |
| Cognition of Arousal (C) | 2 | 52.895 | 3.391 | .039 |
| Sex (D) | 1 | 24.083 | 1.544 | n. s. |
| B X C | 2 | 57.562 | 3.691 | .030 |
| B X D | 1 | 6.750 | < 1 | |
| C X D | 2 | 1.021 | < 1 | |
| B X C X D | 2 | 19.188 | 1.230 | n. s. |
| Error[a] | 72 | 15.599 | | |

[a] Within-cells error from Epinephrine and No Epinephrine cells.

### Table 30b

### Summary of Analysis of Covariance Within No Epinephrine Condition: Effects of Remaining Independent Variables on Subjective Report with Post-Injection Pulse Rate, Systolic Blood Pressure, and Diastolic Blood Pressure as Covariates

| Source | df | MS | F | p |
|---|---|---|---|---|
| Attribution (B) | 1 | 51.523 | 3.598 | .062 |
| Cognition of Arousal (C) | 2 | 51.679 | 3.609 | .032 |
| Sex (D) | 1 | 1.041 | < 1 | |
| B X C | 2 | 46.016 | 3.213 | .046 |
| B X D | 1 | 5.147 | < 1 | |
| C X D | 2 | .668 | < 1 | |
| B X C X D | 2 | 7.610 | < 1 | |
| Within-cells regression[a] | 3 | 44.980 | 3.141 | .031 |
| Error[b] | 69 | 14.317 | | |

[a] Pooled within-cells regression from Epinephrine and No Epinephrine cells.

[b] Within-cells error from Epinephrine and No Epinephrine cells.

Table 31a

Summary of Analysis of Variance Within Epinephrine Condition: Effects of Remaining Independent Variables on Observed Behavior

| Source | df | MS | F | p |
|---|---|---|---|---|
| Attribution (B) | 1 | .521 | $< 1$ | |
| Cognition of Arousal (C) | 2 | .146 | $< 1$ | |
| Sex (D) | 1 | 5.333 | 8.930 | .004 |
| B X C | 2 | .583 | $< 1$ | |
| B X D | 1 | 0.000 | $< 1$ | |
| C X D | 2 | .333 | $< 1$ | |
| B X C X D | 2 | .063 | $< 1$ | |
| Error[a] | 72 | .597 | | |

[a] Within-cells error from Epinephrine and No Epinephrine cells.

Table 31b

Summary of Analysis of Covariance Within Epinephrine Condition: Effects of Remaining Independent Variables on Observed Behavior with Post-Injection Pulse Rate, Systolic Blood Pressure, and Diastolic Blood Pressure as Covariates

| Source | df | MS | F | p |
|---|---|---|---|---|
| Attribution (B) | 1 | .003 | < 1 | |
| Cognition of Arousal (C) | 2 | .524 | < 1 | |
| Sex (D) | 1 | 4.404 | 8.388 | .005 |
| B X C | 2 | .164 | < 1 | |
| B X D | 1 | .024 | < 1 | |
| C X D | 2 | .044 | < 1 | |
| B X C X D | 2 | .034 | < 1 | |
| Within-cells regression[a] | 3 | 2.256 | 4.297 | .008 |
| Error[b] | 69 | .525 | | |

[a] Pooled within-cells regression from Epinephrine and No Epinephrine cells.

[b] Within-cells error from Epinephrine and No Epinephrine cells.

Table 32a

Summary of Analysis of Variance Within No Epinephrine Condition: Effects of Remaining Independent Variables on Observed Behavior

| Source | df | MS | F | p |
|---|---|---|---|---|
| Attribution (B) | 1 | .521 | < 1 | |
| Cognition of Arousal (C) | 2 | .083 | < 1 | |
| Sex (D) | 1 | 2.083 | 3.488 | .066 |
| B X C | 2 | .021 | < 1 | |
| B X D | 1 | .021 | < 1 | |
| C X D | 2 | .146 | < 1 | |
| B X C X D | 2 | 2.333 | 3.907 | .024 |
| Error[a] | 72 | .597 | | |

[a] Within-cells error from Epinephrine and No Epinephrine cells.

Table 32b

Summary of Analysis of Covariance Within No Epinephrine Condition: Effects of Remaining Independent Variables on Observed Behavior with Post-Injection Pulse Rate, Systolic Blood Pressure, and Diastolic Blood Pressure as Covariates

| Source | df | MS | F | p |
|---|---|---|---|---|
| Attribution (B) | 1 | .072 | $< 1$ | |
| Cognition of Arousal (C) | 2 | .073 | $< 1$ | |
| Sex (D) | 1 | .487 | $< 1$ | |
| B X C | 2 | .156 | $< 1$ | |
| B X D | 1 | .005 | $< 1$ | |
| C X D | 2 | .387 | $< 1$ | |
| B X C X D | 2 | 1.200 | 2.285 | n. s. |
| Within-cells regression[a] | 3 | 2.256 | 4.297 | .008 |
| Error[b] | 9 | .525 | | |

[a] Pooled within-cells regression from Epinephrine and No Epinephrine cells.

[b] Within-cells error from Epinephrine and No Epinephrine cells.

# APPENDIX E

# CELL MEANS AND STANDARD DEVIATIONS FOR VARIABLES ANALYZED BY ANOVA, ADJUSTED CELL MEANS FOR VARIABLES ANALYZED BY ANACOVA, AND FREQUENCY PER CELL FOR VARIABLES ANALYZED BY CHI-SQUARE

| Attribution of Arousal | | Extrinsic | | | | | | Intrinsic | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Cognition of Arousal | | No Information | | Unaroused | | Aroused | | No Information | | Unaroused | | Aroused | |
| Subject Sex | | F | M | F | M | F | M | F | M | F | M | F | M |
| Actual Arousal | Epinephrine | 1111 | 1112 | 1121 | 1122 | 1131 | 1132 | 1211 | 1212 | 1221 | 1222 | 1231 | 1232 |
| | No Epinephrine | 2111 | 2112 | 2121 | 2122 | 2131 | 2132 | 2211 | 2212 | 2221 | 2222 | 2231 | 2232 |

Figure 2. Schematic Representation of the Design and Cell Codes.

### Table 33

### Cell Means and Standard Deviations

| Cell | | Subjective Report | Autonomic Report | Observed Behavior | Severity | Amount of Anesthetic[a] | Pre-Injection Pulse Rate | Post-Injection Pulse Rate |
|---|---|---|---|---|---|---|---|---|
| 1111 | M | 2.500 | 3.250 | 1.625 | 2.250 | 1.125 | 79.000 | 88.000 |
| | SD | 5.802 | 1.708 | 1.109 | 0.957 | 0.250 | 10.000 | 12.649 |
| 1112 | M | -3.750 | 1.000 | 1.000 | 2.250 | 1.750 | 82.000 | 91.000 |
| | SD | 5.123 | 1.414 | 0.707 | 0.500 | 0.289 | 18.037 | 19.967 |
| 1121 | M | -1.750 | 2.000 | 1.375 | 1.500 | 1.400 | 76.000 | 80.000 |
| | SD | 4.992 | 0.816 | 0.479 | 0.577 | 0.424 | 8.641 | 7.303 |
| 1122 | M | -0.750 | 2.500 | 0.875 | 1.750 | 1.200 | 69.000 | 79.000 |
| | SD | 3.500 | 0.577 | 0.750 | 0.957 | 0.245 | 11.944 | 6.831 |
| 1131 | M | 5.000 | 3.500 | 1.500 | 2.000 | 1.200 | 87.000 | 90.000 |
| | SD | 2.160 | 2.646 | 1.000 | 0.816 | 0.245 | 10.520 | 7.659 |
| 1132 | M | -3.250 | 1.750 | 0.625 | 1.750 | 1.150 | 61.000 | 69.000 |
| | SD | 3.775 | 2.062 | 0.479 | 0.500 | 0.173 | 13.216 | 9.452 |
| 1211 | M | -1.500 | 2.250 | 1.375 | 2.000 | 1.375 | 85.000 | 90.000 |
| | SD | 5.745 | 2.217 | 0.750 | 0.816 | 0.479 | 13.614 | 20.785 |
| 1212 | M | 1.750 | 1.250 | 1.000 | 3.500 | 1.875 | 77.500 | 84.000 |
| | SD | 1.258 | 1.258 | 0.816 | 2.517 | 0.250 | 5.000 | 5.657 |
| 1221 | M | -3.000 | 3.500 | 1.500 | 1.500 | 1.250 | 83.000 | 83.000 |
| | SD | 5.354 | 4.123 | 0.408 | 1.000 | 0.500 | 13.614 | 5.033 |
| 1222 | M | 0.750 | 2.000 | 1.000 | 2.500 | 1.375 | 83.000 | 95.000 |
| | SD | 1.893 | 1.155 | 0.408 | 1.732 | 0.479 | 11.944 | 10.000 |
| 1231 | M | 7.000 | 6.750 | 2.250 | 4.000 | 1.625 | 105.000 | 110.000 |
| | SD | 1.826 | 1.500 | 0.866 | 2.160 | 0.479 | 18.294 | 20.785 |
| 1232 | M | 2.500 | 2.750 | 1.125 | 1.750 | 1.000 | 74.000 | 75.000 |
| | SD | 2.646 | 0.957 | 0.479 | 1.500 | 0.000 | 20.265 | 15.449 |

Table 33 (continued)

| Cell | | Subjective Report | Autonomic Report | Observed Behavior | Severity | Amount of Anesthetic[a] | Pre-Injection Pulse Rate | Post-Injection Pulse Rate |
|---|---|---|---|---|---|---|---|---|
| 2111 | M | 3.000 | 1.250 | 1.625 | 2.250 | 1.125 | 96.000 | 96.000 |
| | SD | 3.367 | 1.258 | 1.109 | 1.258 | 0.250 | 10.832 | 9.798 |
| 2112 | M | -0.500 | 1.250 | 0.625 | 2.750 | 1.500 | 77.000 | 74.000 |
| | SD | 2.887 | 2.500 | 0.479 | 2.363 | 0.707 | 6.831 | 5.164 |
| 2121 | M | -1.000 | 0.250 | 1.000 | 1.250 | 1.000 | 61.000 | 59.000 |
| | SD | 2.944 | 0.500 | 1.000 | 0.500 | 0.000 | 8.869 | 10.520 |
| 2122 | M | 0.750 | 1.000 | 1.250 | 2.500 | 1.125 | 72.000 | 74.000 |
| | SD | 2.217 | 1.155 | 1.041 | 3.000 | 0.250 | 8.641 | 10.066 |
| 2131 | M | 0.250 | 2.250 | 1.250 | 1.750 | 1.125 | 93.000 | 89.000 |
| | SD | 2.062 | 0.957 | 0.866 | 0.500 | 0.250 | 18.294 | 17.397 |
| 2132 | M | 0.000 | 5.250 | 0.875 | 1.500 | 1.375 | 71.000 | 71.000 |
| | SD | 6.633 | 5.560 | 0.854 | 0.577 | 0.479 | 11.489 | 8.246 |
| 2211 | M | -2.000 | 0.750 | 1.250 | 2.000 | 1.875 | 86.000 | 83.000 |
| | SD | 5.099 | 0.957 | 0.957 | 0.816 | 0.854 | 16.492 | 15.100 |
| 2212 | M | -2.500 | 1.750 | 1.500 | 1.000 | 1.000 | 63.000 | 70.000 |
| | SD | 2.887 | 2.872 | 0.707 | 0.000 | 0.000 | 8.246 | 9.522 |
| 2221 | M | -2.000 | 0.500 | 2.125 | 1.750 | 1.125 | 93.000 | 94.000 |
| | SD | 4.830 | 1.000 | 0.629 | 1.500 | 0.250 | 10.520 | 6.928 |
| 2222 | M | -6.000 | 0.250 | 0.625 | 1.750 | 1.125 | 78.000 | 77.000 |
| | SD | 5.354 | 0.500 | 0.479 | 0.500 | 0.250 | 2.309 | 3.830 |
| 2231 | M | 4.000 | 2.000 | 1.250 | 2.500 | 1.125 | 86.000 | 87.000 |
| | SD | 1.826 | 2.160 | 0.645 | 1.000 | 0.250 | 11.547 | 11.489 |
| 2232 | M | 2.000 | 2.000 | 1.125 | 2.750 | 1.125 | 76.000 | 77.000 |
| | SD | 3.162 | 2.708 | 0.750 | 2.872 | 0.250 | 21.417 | 22.716 |

Table 33 (continued)

| Cell | | Post-Manipulation Pulse Rate | Pre-Injection Systolic Blood Pressure | Post-Injection Systolic Blood Pressure | Post-Manipulation Systolic Blood Pressure | Pre-Injection Diastolic Blood Pressure | Post-Injection Diastolic Blood Pressure | Post-Manipulation Diastolic Blood Pressure |
|---|---|---|---|---|---|---|---|---|
| 1111 | M | 88.000 | 113.500 | 112.500 | 109.500 | 64.500 | 62.000 | 63.000 |
| | SD | 16.330 | 20.744 | 20.615 | 18.646 | 5.972 | 5.888 | 5.773 |
| 1112 | M | 87.000 | 126.000 | 119.500 | 116.500 | 71.500 | 57.000 | 57.000 |
| | SD | 12.383 | 12.000 | 12.583 | 9.147 | 10.755 | 18.655 | 12.910 |
| 1121 | M | 81.000 | 116.000 | 117.000 | 114.250 | 73.000 | 71.000 | 68.500 |
| | SD | 5.033 | 9.381 | 19.356 | 15.586 | 5.291 | 4.761 | 4.435 |
| 1122 | M | 76.000 | 115.000 | 121.000 | 119.500 | 78.500 | 69.000 | 69.000 |
| | SD | 7.303 | 14.742 | 11.605 | 12.369 | 3.416 | 9.018 | 5.292 |
| 1131 | M | 89.000 | 125.000 | 123.000 | 123.500 | 88.500 | 81.500 | 84.000 |
| | SD | 8.246 | 14.095 | 14.468 | 13.601 | 8.062 | 9.000 | 9.092 |
| 1132 | M | 66.000 | 110.500 | 114.000 | 116.000 | 78.000 | 72.500 | 69.500 |
| | SD | 10.583 | 7.895 | 2.828 | 4.321 | 8.165 | 8.544 | 5.745 |
| 1211 | M | 88.000 | 117.500 | 116.500 | 112.500 | 77.000 | 74.000 | 70.500 |
| | SD | 19.322 | 12.689 | 11.475 | 10.376 | 14.468 | 9.381 | 10.247 |
| 1212 | M | 87.000 | 122.000 | 122.500 | 125.000 | 60.500 | 59.000 | 54.000 |
| | SD | 8.869 | 14.787 | 14.731 | 15.100 | 27.295 | 27.055 | 23.944 |
| 1221 | M | 82.000 | 116.000 | 119.000 | 117.000 | 75.000 | 62.500 | 69.000 |
| | SD | 10.066 | 1.633 | 6.218 | 3.464 | 12.275 | 11.818 | 11.136 |
| 1222 | M | 93.000 | 125.000 | 129.000 | 126.500 | 75.500 | 67.000 | 67.500 |
| | SD | 8.869 | 9.592 | 15.011 | 13.000 | 4.435 | 3.464 | 6.191 |
| 1231 | M | 105.000 | 121.000 | 121.000 | 120.000 | 70.000 | 65.500 | 69.000 |
| | SD | 13.614 | 6.831 | 3.464 | 2.828 | 9.933 | 7.188 | 7.024 |
| 1232 | M | 79.000 | 136.500 | 135.500 | 135.000 | 83.500 | 79.000 | 73.500 |
| | SD | 18.294 | 4.123 | 3.416 | 3.830 | 12.897 | 4.761 | 4.435 |

Table 33 (continued)

| Cell | | Post-Manipulation Pulse Rate | Pre-Injection Systolic Blood Pressure | Post-Injection Systolic Blood Pressure | Post-Manipulation Systolic Blood Pressure | Pre-Injection Diastolic Blood Pressure | Post-Injection Diastolic Blood Pressure | Post-Manipulation Diastolic Blood Pressure |
|---|---|---|---|---|---|---|---|---|
| 2111 | M | 91.000 | 120.000 | 116.500 | 116.500 | 75.500 | 73.000 | 72.000 |
| | SD | 6.000 | 9.522 | 7.550 | 8.544 | 4.435 | 6.831 | 9.381 |
| 2112 | M | 73.000 | 129.000 | 129.500 | 127.500 | 83.000 | 81.500 | 84.500 |
| | SD | 6.831 | 8.083 | 14.457 | 13.796 | 3.464 | 13.699 | 9.983 |
| 2121 | M | 64.000 | 110.000 | 109.500 | 114.000 | 69.000 | 66.000 | 67.500 |
| | SD | 9.798 | 20.461 | 19.485 | 19.114 | 12.275 | 9.092 | 6.608 |
| 2122 | M | 75.000 | 121.500 | 123.000 | 123.000 | 73.500 | 72.500 | 70.500 |
| | SD | 8.869 | 11.475 | 15.874 | 16.693 | 9.147 | 8.062 | 6.807 |
| 2131 | M | 90.000 | 126.500 | 129.000 | 127.500 | 78.500 | 82.000 | 81.500 |
| | SD | 14.787 | 16.603 | 14.652 | 16.442 | 12.793 | 15.748 | 12.152 |
| 2132 | M | 75.000 | 131.000 | 130.000 | 129.000 | 86.500 | 86.000 | 85.000 |
| | SD | 8.869 | 5.774 | 2.828 | 3.464 | 13.601 | 14.329 | 11.605 |
| 2211 | M | 86.000 | 130.500 | 129.000 | 125.000 | 79.500 | 76.500 | 80.000 |
| | SD | 16.492 | 8.699 | 9.019 | 14.095 | 10.878 | 15.177 | 10.456 |
| 2212 | M | 69.000 | 129.500 | 129.000 | 128.000 | 73.000 | 77.500 | 78.000 |
| | SD | 11.944 | 14.177 | 20.298 | 20.199 | 11.832 | 10.376 | 12.111 |
| 2221 | M | 94.000 | 115.500 | 113.000 | 115.000 | 74.000 | 69.000 | 72.500 |
| | SD | 9.522 | 10.630 | 9.019 | 10.132 | 12.111 | 13.115 | 12.897 |
| 2222 | M | 76.000 | 124.500 | 126.000 | 125.500 | 79.500 | 77.000 | 80.000 |
| | SD | 8.641 | 5.260 | 5.888 | 5.508 | 7.724 | 15.875 | 12.329 |
| 2231 | M | 85.000 | 112.000 | 115.500 | 113.000 | 73.000 | 70.500 | 68.500 |
| | SD | 8.869 | 6.928 | 6.608 | 5.033 | 7.746 | 4.435 | 5.972 |
| 2232 | M | 77.000 | 121.500 | 122.000 | 121.000 | 77.500 | 71.500 | 75.500 |
| | SD | 19.425 | 13.404 | 13.367 | 14.652 | 10.247 | 9.983 | 11.121 |

[a] Number of cartridges.

Table 34

Cell Mean Estimates for Variables Adjusted Within Epinephrine Condition

| Cell | Post-Injection Pulse Rate Adjusted for Pre-Injection Pulse Rate | Post-Injection Pulse Rate Adjusted for Pre-Injection Pulse Rate and Amount of Anesthetic | Subjective Report Adjusted for Amount of Anesthetic |
|---|---|---|---|
| 1111 | 88.936 | 88.588 | 1.583 |
| 1112 | 89.440 | 90.017 | -2.232 |
| 1121 | 83.432 | 83.414 | -1.596 |
| 1122 | 88.255 | 87.836 | -1.375 |
| 1131 | 84.280 | 84.177 | 4.375 |
| 1132 | 84.911 | 84.281 | -4.070 |
| 1211 | 85.944 | 86.050 | -1.443 |
| 1212 | 86.184 | 86.856 | 3.755 |
| 1221 | 80.608 | 80.505 | -3.430 |
| 1222 | 92.608 | 92.679 | 0.807 |
| 1231 | 89.305 | 90.112 | 8.031 |
| 1232 | 80.096 | 79.485 | 1.096 |

## Table 35

### Cell Mean Estimates for Autonomic Measures Adjusted for Previous Level on the Same Measure

| Cell | Post-Injection Pulse Rate | Post-Manipulation Pulse Rate | Post-Injection Systolic Blood Pressure | Post-Manipulation Systolic Blood Pressure | Post-Injection Diastolic Blood Pressure | Post-Manipulation Diastolic Blood Pressure |
|---|---|---|---|---|---|---|
| 1111 | 88.603 | 83.560 | 120.172 | 117.976 | 70.807 | 70.272 |
| 1112 | 89.123 | 80.043 | 115.153 | 118.576 | 60.327 | 67.986 |
| 1121 | 83.083 | 83.272 | 122.268 | 118.612 | 73.153 | 69.088 |
| 1122 | 87.869 | 79.111 | 127.229 | 120.205 | 66.847 | 71.073 |
| 1131 | 83.990 | 82.882 | 119.615 | 122.376 | 71.518 | 76.790 |
| 1132 | 84.482 | 77.501 | 124.556 | 123.104 | 70.739 | 68.974 |
| 1211 | 85.643 | 81.882 | 120.326 | 117.319 | 73.021 | 68.860 |
| 1212 | 85.843 | 85.916 | 121.999 | 124.333 | 70.939 | 63.500 |
| 1221 | 80.296 | 81.755 | 124.268 | 119.533 | 63.087 | 75.901 |
| 1222 | 92.296 | 82.687 | 125.615 | 119.891 | 67.196 | 71.059 |
| 1231 | 89.110 | 82.102 | 121.461 | 120.705 | 70.001 | 73.673 |
| 1232 | 79.736 | 85.467 | 121.058 | 122.448 | 72.933 | 68.146 |
| 2111 | 82.550 | 79.848 | 117.922 | 121.319 | 73.196 | 71.103 |
| 2112 | 76.256 | 80.306 | 122.269 | 120.434 | 75.824 | 77.290 |
| 2121 | 74.482 | 83.891 | 120.537 | 125.218 | 71.284 | 71.801 |
| 2122 | 80.389 | 82.306 | 122.980 | 121.876 | 74.261 | 69.974 |
| 2131 | 78.030 | 84.721 | 124.173 | 120.891 | 79.847 | 73.918 |
| 2132 | 78.216 | 84.823 | 120.846 | 121.477 | 77.584 | 74.447 |
| 2211 | 77.816 | 85.755 | 120.327 | 118.391 | 73.564 | 76.503 |
| 2212 | 83.829 | 79.662 | 121.288 | 121.391 | 79,653 | 73.760 |
| 2221 | 83.030 | 84.526 | 118.749 | 123.019 | 70.370 | 74.573 |
| 2222 | 78.429 | 80.789 | 123.096 | 121.634 | 74.064 | 76.132 |
| 2231 | 81.816 | 81.399 | 124.614 | 118.733 | 72.653 | 69.459 |
| 2232 | 80.083 | 81.789 | 121.980 | 120.790 | 70.130 | 75.717 |

## Table 36

Cell Mean Estimates for Variables Adjusted for Post-Injection Pulse Rate, Systolic Blood Pressure, and Diastolic Blood Pressure

| Cell | Subjective Report | Observed Behavior |
|---|---|---|
| 1111 | 1.423 | 1.426 |
| 1112 | -5.710 | 0.572 |
| 1121 | -1.485 | 1.451 |
| 1122 | -0.697 | 0.881 |
| 1131 | 5.295 | 1.583 |
| 1132 | -1.905 | 0.939 |
| 1211 | -1.728 | 1.363 |
| 1212 | 0.445 | 0.694 |
| 1221 | -3.810 | 1.323 |
| 1222 | -0.848 | 0.626 |
| 1231 | 4.298 | 1.690 |
| 1232 | 3.410 | 1.259 |
| 2111 | 2.209 | 1.497 |
| 2112 | 0.878 | 0.892 |
| 2121 | 0.646 | 1.378 |
| 2122 | 1.466 | 1.390 |
| 2131 | 0.512 | 1.296 |
| 2132 | 2.017 | 1.282 |
| 2211 | -1.777 | 1.272 |
| 2212 | -1.166 | 1.749 |
| 2221 | -2.913 | 1.978 |
| 2222 | -5.182 | 0.784 |
| 2231 | 3.709 | 1.220 |
| 2232 | 2.415 | 1.206 |

Table 37

Total Number of Cases in Each Cell for Variables Tested by Chi-Square

| Cell | Avoid[a] | Dental Attribution | Drug Attribution |
|---|---|---|---|
| 1111 | 3 | 2 (4)[b] | 2 |
| 1112 | 1 | 1 (2) | 1 |
| 1121 | 0 | 4 (4) | 1 |
| 1122 | 1 | 2 (4) | 3 |
| 1131 | 2 | 4 (4) | 1 |
| 1132 | 1 | 1 (2) | 1 |
| 1211 | 2 | 3 (3) | 1 |
| 1212 | 2 | 2 (3) | 0 |
| 1221 | 1 | 1 (2) | 1 |
| 1222 | 1 | 3 (4) | 0 |
| 1231 | 2 | 4 (4) | 1 |
| 1232 | 3 | 3 (4) | 0 |
| 2111 | 3 | 3 (3) | 1 |
| 2112 | 0 | 1 (1) | 1 |
| 2121 | 3 | 1 (1) | 0 |
| 2122 | 2 | 2 (2) | 0 |
| 2131 | 2 | 4 (4) | 1 |
| 2132 | 2 | 3 (3) | 3 |
| 2211 | 1 | 2 (2) | 0 |
| 2212 | 1 | 2 (2) | 1 |
| 2221 | 1 | 1 (1) | 0 |
| 2222 | 0 | 1 (1) | 0 |
| 2231 | 1 | 3 (3) | 0 |
| 2232 | 1 | 2 (3) | 0 |

[a] The number of subjects predicting Return was 4 minus the number predicting Avoid.

[b] The number in the parentheses is the number of subjects in the cell who reported arousal and were consequently asked for their attributions.

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